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INTERNATIONAL PHYTOPATHOLOGIC COLLABORATION

WORK BEGUN IN EUROPE—WILL IT BE PROSECUTED IN AMERICA?

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THE BEARING OF THE QUESTION

During the last twenty-five years we have seen in Europe an endeavor to bring together the phytopathologists of different countries to a systematic collaboration for combating the diseases of cultivated plants.

This idea is based on the fact, now, as I think, uncontested, that not only the growers but also the men of science are in most cases puzzled as to how to act against the parasites of different kinds, which now and then menace the culture of plants. Why this annoying state of things? The answer to this question is that our impotence against these enemies is due chiefly to serious deficiencies in our knowledge of the nature of the destroyers.

In a few cases only, do we really know the nature of these so well, that we may found on this knowledge an effective control of the diseases. Such is the case with the smuts (Tilletia and Ustilago) on the cereals, parasites that we know well enough to be able somewhat to control.

But in how many other cases is this true?

The common potato disease (*Phytophthora infestans*), known in Europe and in North America since about the year 1845, has been diligently and repeatedly studied on both continents. In spite of all efforts the parasite has spread to all countries where potatoes are cultivated, at last (1904) to Australia. It is true that we have learned that the spraying of the potato field with fungicides is an effective method of restricting the destructive effects of the fungus. Further we profit by the fact that varieties of potatoes differ in their susceptibility to attack by the parasite. But we are not advanced any further. We are yet incapable of preventing an outbreak of the disease or of stopping the spread of it. In spite

of all researches, even the latest, the wintering of the fungus from year to year is yet an unsolved problem.

The grain rusts (*Puccinia graminis, etc.*) are known from ancient time as causing very destructive epidemics in all corn growing countries. By new and profound studies our knowledge of the nature of the parasites is extended, especially in reference to their polymorphism and to their spread by means of infected seed. No unanimous conception of the results however has been acquired and consequently no commonly accepted basis given for extensive practical efforts to combat the diseases. Last year our hopes lay in one of the great European states, that seemed inclined to start a new profound investigation on the grain rusts. Unfortunately the great war began, overthrowing all these plans.

The gooseberry mildew (*Sphaerotheca mors-uvae*) is a new disease to Europe. The first appearance of the parasite in Europe (Ireland, Denmark) was reported in the year 1900. In the course of ten years it occurred in all the European countries in which gooseberries are cultivated. As a consequence of this the authorities were driven to the necessity of taking their refuge in legislative measures. Quarantines were established against the transportation of gooseberry plants within the countries as well as against the importation of foreign gooseberry plants and fruits. Arrangements for inspection and control of infected gardens were made, and especially in England, a vast and expensive service was organized. The cultivators tried spraying with liquid fungicides. Unfortunately it had escaped the memory or the attention of the authorities and of the scientists, that there are important blanks in our knowledge of the biology of the parasite. The life-history of the fungus had not been followed throughout the whole year. The germination of the ascospores and the infection by means of these spores are as yet not cleared up. The occasional, sudden outbreak of the disease in the height of summer (July) is also unexplained. If all these questions had been clear at the time of the immigration of the pest into Europe, great sums spent on fruitless labor would have been spared; and by the employment of effective means gooseberry culture—the most important berry-culture of North and Middle Europe—would have been saved from a seriously impending ruin.

LOSSES CALCULATED

In different countries calculations have been made of the losses caused by the diseases of cultivated plants. Astounding figures have resulted. Through the grain-rusts, Germany lost in the year of 1891 about $100,000,000 and the United States of North America, through the rust of wheat, about $67,000,000. The yearly loss caused to the world crop by the

grain-rusts is estimated at about $250,000,000. The annual loss of the United States of North America through the potato blight is estimated at about $36,000,000 and so on. The powdery mildew of the grape (*Oidium Tuckeri*) at first noticed in Europe (England), about 1845, is said to have reached every European vineyard before 1851. The downy mildew of the grape (*Plasmopara viticola*) made its first appearance in Europe in 1870 and within ten years reached all grape-growing countries of that continent. In 1895, this parasite caused in Hungary a loss of about 12,000,000 of hectrolitres of wine. The annual loss caused by the diseases to the grape crop of the whole world is estimated at about $2,500,000,000.

THE QUESTION BEFORE CONGRESSES

The idea of an international collaboration in order to control the most destructive diseases of cultivated plants was made public for the first time, in Vienna in 1890. Later the same question was presented to international meetings: 1900 in Paris; 1903, 1905 and 1907 in Rome; 1907 in Vienna; 1908 in Montpellier; 1909 and 1910 in Rome; 1912 in Paris; 1913 and 1914 in Rome.

All these conferences have insisted on the need of genuine collaboration between the pathologists of the different countries in order to acquire a fuller knowledge as to the nature and the evolution of the parasites, with a view of finding out new methods for combating the enemies.

ROME AS CENTRE OF THE COLLABORATION

The establishment of the International Institute of Agriculture at Rome being decided, thoughts were turned towards this Institute as the centre of the collaboration. Plans for the organization of the work according to these ideas were presented to several general assemblies of the Institute. At first the Institute took a fairly adverse position on the question, which was out of its program. The prerequisites for the realization of the plans —especially money and staffs—were lacking. But gradually the position of the Institute towards the question grew more favorable, especially from the time of the International Pathological Congress at Paris in 1912. This Congress pronounced itself in favor of it. The consequence of this turning was a request addressed by the General Assembly at Rome in 1913, to the French Government to invite all contributing states of the Institute to an International Phytopathological Conference at Rome. Such a Conference was held there in 1914 from February 25 to March 4. At that Conference were present 59 delegates from 31 states, of which 20 were European and 11 extra-European.

INTERNATIONAL PHYTOPATHOLOGICAL CONVENTION

The chief aim of the Conference was to organize an International Phytopathological Convention, acceptable to all states. This Convention should establish the principles for the organization of a Phytopathological service in different countries. After a long deliberation an *Acte final de la Conference Internationale de Phytopathologie, fait à Rome le 4 Mars 1914*, was accepted by the delegates. This act which contained twenty-one different articles, with forms of certificates, was to be submitted to the governments of the different states for consideration and approval.

It must be admitted freely that through the decisions of this Conference the question of an international collaboration in order to combat the diseases of plants is in some degree solved. By an official professional inspection and control the principles are fixed for preventing, as far as possible, the spread of dangerous diseases from culture to culture, from land to land.

SCIENTIFIC RESEARCHES NOT ADVANCED

However the struggle against the diseases of plants comprehends much more than inspection and control. There is also another matter—and this I think very important—that is the gaining of more effective means for combating the diseases. For reaching of such means no inspection or control legislation will help. The only way to that end is continued research and continued experiments, in other words, a strengthening and development of scientific pathological work.

In a series of papers I have, since 1890, at several international congresses, drawn attention to this fact. Of late I have done it by the report *Les problémes phytopathologiques. Les différences des maladies exigent des mesures différentes*, presented to Conference at Rome in 1914.

From a careful consideration it will be obvious that this side of the phytopathological question is by no means forwarded through the arrangements made in Europe hitherto. Rather a regression of phytopathological research is to be feared. In several states, where a phytopathological service has been introduced, for instance in Holland and in Italy, this service is located at existing experimental stations. The original and specific task of those stations, investigation and experimentation, must by this arrangement be seriously hindered. Misgivings in that direction are also expressed from different countries. Such being the case, I took the liberty, after having conferred with several colleagues, at the final session of the Conference, to say an open word of warning against the danger here threatening. The want is not supplied by enlarged staffs and increased grants to those stations. The phytopathological inspection

service is of a regulatory character, and ought to be organized separately and placed under administrative authority. The services of the scientific experimental workers may not be required, except in unusual cases, that is, if the competency of the service is insufficient.

THE GREAT WAR AND THE FUTURE OF THE COLLABORATION IN EUROPE

The continuation of an international phytopathological collaboration in Europe is rendered impossible, I think, for a long time to come through the great war that broke out last year. The harmony between the nations of Europe is now so deranged that the reestablishment of peace will probably not be able to heal the wounds. The common work is doomed in Europe, certainly for decades.

NORTH AMERICA IN THE PLACE OF EUROPE

Shall this work cease? I do not believe it. There is a continent greater than all the states of Europe combined, which is out of the war. This continent has the most varying natural conditions and kinds of soil and has at its disposal all the resources of money and of scientists required for undertaking the work. That land is the great continent of North America, in the first place the United States.

Judging from remarkable utterances made by professionals of distinction in the American press, the soil seems to be well prepared and suitable for the transportation of the tender, languishing plant of collaboration from European to American ground. Numerous articles by American phytopathologists for the last decennium give evidence of a more and more awakened interest in phytopathological research.

If the United States of North America is willing to take the initiative and the charge of an organized collaboration against the diseases of our cultivated plants, that now cause colossal losses to all countries of the world, they will further vindicate their place of rank in the science and they will deserve well of the gratitude of all mankind.

STOCKHOLM
SWEDEN

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1890–1914

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LEAF ROLL DISEASES OF THE POTATO[1]

DR. O. APPEL

The importance of the potato as a cultural crop has led many phytopathologists to devote their attention to the diseases of this plant. Many of the problems involved are complex and much remains to be done before a satisfactory solution is reached, but the progress so far made is such as to justify our presenting a résumé of what has been accomplished and calling attention to the problems which it is highly desirable to solve.

All potato diseases can not, of course, be discussed in a single lecture, consequently I will restrict my remarks to the group which seems to me the most interesting, that is the group which is characterized by a rolling of the leaves.

From the literature on the subject you know that in former years tremendous epidemics occurred among potatoes, the cause of which was assumed to be the disease called "curly dwarf." Examination of the extensive literature concerning these epidemics shows that the numerous descriptions of the disease are by no means concordant, and therefore there is hardly any doubt that the different authors were concerned with different diseases. I have tried to bring some order out of this chaos by dividing the "curly dwarf" of the old literature into three types, which I have designated as the curly dwarf (in a more restricted sense), the leaf roll disease, and the bacterial ring disease. I have at the same time pointed out that in all probability another division of the leaf roll disease would be necessary, and this has actually proved to be the case, for it has developed that the disease which I originally designated as the leaf roll disease was in reality a combination of several diseases.

Investigation of the cause of the leaf-roll symptoms shows that it is a disturbance of the water balance of the plant. If a plant transpires more water than can be supplied, as often happens during very hot days, it wilts. If transpiration constantly exceeds absorption to some degree the leaves do not expand fully, but fold parallel to their longitudinal axis so that either the two halves of the leaf finally lie against one another or the leaf margins roll in toward the midrib. Such a rolling may be caused by various influences, such as external conditions which affect the root system, internal conditions which influence the chemical composition of the

[1] A lecture delivered at the Universities of Iowa, Minnesota, Wisconsin and Cornell.

plant, or the disorganization of the vascular system. Parasites also may have a pernicious influence on the water balance by living in the vessels, thus distorting the normal transport of water and nutrient substances, or they destroy the tissues completely.

As the symptoms of diseases differ so do their causes, and from external symptoms it is often difficult to determine the disease with which we have to deal. It should never be forgotten, however, that a sure diagnosis can be made only on the basis of thorough investigation.

Let us now consider the different aspects of the diseases, beginning with the true curly dwarf (Kräuselkrankheit), which name I use to designate all diseases of the vine that show shortened internodes and curly leaves. The curly appearance is caused by a shortening of the midribs. The leaf lamina is larger than the space between the different ribs, consequently the leaf becomes undulate. The stems of such plants are exceedingly brittle and very often the leaves remain small. When the disease is very distinct the plants remind us of winter cabbage.

Potato vines affected with curly dwarf give small yields and produce small tubers. The next year these tubers produce diseased vines. In most cases no normal tubers are produced by these vines and consequently are not used for seed purposes, which fact prevents wide dissemination of the disease. Curly dwarf has never been widespread and I have not found an epidemic of the disease the last fifteen years, and likewise in the United States I have found it occurring only sporadically. It is only within the limits of experiments with certain varieties that occasionally all vines are diseased. Sometimes plant breeders find distinct varieties completely affected, but these are eliminated and never find their way into agriculture.

The disease called "streak" in America is in some respects similar to curly dwarf. The symptoms of the disease are black streaks on the stems and leaf nerves, which are frequently accompanied by black spots on the leaves. As early as 1897 Frank described this disease, designating it under the collective name of "vine diseases" (Staudenkrankheiten). In my work entitled Die Blattrollkrankheit und unsere Kartoffelernten, published in collaboration with my assistant Dr. Schlumberger, I have shown that the vine disease of Frank includes curly dwarf as well as streak and that it is also a combination of these two diseases.

The streak disease seems to be of bacterial origin, but so far its real cause has not been discovered. In cases where it is but weakly developed it does not resemble curly dwarf, but sometimes the internodes are shortened and the leaves curled, and in these it is always apparent that the black streaks and the curling and dwarfed appearance are due to the same cause. These injuries cause a decrease in the yield, but whether the disease is transmitted through the seed has not been proved. The

trouble is pretty common in the United States and it would be a good thing to investigate it.

The next disease about which I wish to speak is leaf-roll. In this disease the rolling of the leaves is most distinct and is one of its most reliable diagnostic characters. This phenomenon consists in the upward and inward rolling, more or less parallel to the midrib, of the individual leaflets. Sometimes this rolling is more pronounced at the base of the leaflet, the result in such case being a somewhat funnel-shaped structure. No undulation of the leaf surface or of the leaf margin accompanies this rolling. In seriously diseased or inadequately nourished plants the rolling occurs especially early and is very pronounced, while in plants less seriously affected or well nourished it is slow to make its appearance. Coincident with the rolling a discoloration, which may be either yellowish green or reddish, depending on the variety of potato, frequently develops. In some varieties the discoloration is almost violet. The intensity of the discoloration varies in different years, but appears to be more pronounced in dry years than in wet ones.

A further characteristic of seriously diseased plants is the peculiar manner of growth. In such plants the vines are shorter and the leaves assume a more erect position, and consequently before flowering have a more rigid appearance than similar healthy plants. Here again, however, the individual varieties differ. Another diagnostic character of diseased plants is the smallness of the flowers, leaves, and berries as compared with those of healthy plants.

In the case of seriously diseased plants there is frequently little or no tuber production. Such plants show either a very poor development with no tubers and die early or a good development of vines, which remain green until toward the end of the vegetative period, but which usually produce only very small tubers, the latter developing on foreshortened stolons or even directly on the subterranean stem end. The latter characteristic, however, is not constant, as frequently plants occur with normally developed stolons which bear a number of tubers of the most varying size. Although slightly affected plants may produce a large yield the plants grown from such tubers are certain to give only a very small yield.

Longevity of the seed tubers is characteristic of the disease. In many cases, though not in all, the diseased seed tubers remain alive until the crop is harvested. When such tubers are found the case is suspicious. One of the most distinct peculiarities of the disease is its inheritance, if I may so call it, by the seed potato, which in turn produces diseased plants. The inheritance through the seed has often been observed. In severe cases of the disease the symptoms are very distinct and the diagnosis consequently easy, but in many cases, such for instance as normal-

sized vines where the plants are characterized only by the rolling of the leaves, it is very difficult to recognize the leaf roll as such. With a little training, however, leaf roll may be distinguished from other diseases. Besides external symptoms it has an anatomical characteristic. Quanjer[2] has found that the phloem of the diseased plant is different from that of the healthy one, being pressed together and having an abnormal structure. For diagnostic purposes, however, this symptom should be used with precaution, for Schander[3] discovered similar peculiarities in normal but old plants. The vessels do not show any change of structure.

The phases of sprouting of diseased tubers are peculiar. The digestion of reserve food materials in the diseased tubers is a much slower process than in healthy potatoes. It is possible that this depends upon a quantitative difference of the enzymes, but the investigations published up to this time have not thrown sufficient light on this question.

Necrosis of the phloem and change in the enzymes may be the cause of the external symptoms, but these are not the cause of the disease itself. The cause has not yet been found, but it is clear that the mycelium of Fusarium, which often occurs in the tissues, has nothing whatever to do with it. Climatic and soil conditions influence the intensity of the disease, but can not be its cause, although there are localities which evidently favor it.

The opinion of Hedlund[4] that the leaf roll is a diseased mutation has some plausibility, but is only an attempt at explication and is not supported by any proofs. The sudden and general appearance of the disease can not be explained in this way.

The disease is of great economic importance as it seriously reduces the yields. In Colorado, for instance, it reduced the crop from some fields to one-fourth what it should be. In the western part of Germany this disease has been of much importance, but by means of field inspection it has been practically overcome, so that now it has no influence on the total crop of the Empire. I will discuss this field inspection and seed certification later on.

Other diseases of this group which might be mistaken for the leaf roll are the so-called "vascular mycoses." As already stated, the rolling of leaves is the result of disturbance in the water balance. So far as known,

[2] Quanjer, H. M. Die Nekrose des Phloëms der Kartoffelpflanze als Ursache der Blattrollkrankheit. Mededeelingen, Rijks Hoogere Land-, Tuin- en Boschbouwschool, Deel VI, 1913.

[3] Schander, R., und von Tiessenhausen, M. Kann man dis Phloëmnekrose als Ursache oder Symptom der Blattrollkrankheit der Kartoffel ansehen. Mitteilungen des Kaiser Wilhelm Instituts für Landwirtschaft in Bromberg, **6**: 115. 1914.

[4] Hedlund, T. Nagra Jakttagelser öfver bladrullsjuka hospotatis. Tidskrift förLandtmän, 31 Sta. Arg., 1910.

the disturbance in this case is caused by fungi of the genera Fusarium and Verticillium. As these diseases also produce a rolling of the leaves I did not at first distinguish them from leaf roll and I described the leaf roll as being of a parasitic character. Only later on was it proved that two diseases, one of a parasitic and the other of a non-parasitic nature, existed under the name of leaf roll. The early diagnosis of leaf roll disease includes the symptoms of both.

In the non-parasitic leaf roll there is no fungus in the wood vessels and no discoloration. In the parasitic leaf roll, tubers in which there is no fungus mycelium do not transmit the disease.

I wish to eliminate the name of leaf roll disease for the parasitic troubles and substitute the name vascular mycosis. The Austrian pathologists Kornauth, Köch, Himmelbauer, et al., adhere to the former term for these troubles and this must be borne in mind in dealing with Austrian literature on the subject.

The wilt disease described by Erwin F. Smith and D. B. Swingle may be taken as a prototype of the vascular mycoses. This wilt is widespread in the United States. On my trip I found it in every state visited. In some fields as high as 70 to 80 per cent of the plants were affected, something which I had never witnessed before. The disease occurs in Germany also, but is of much less importance.

There is some difference between the rolling of the leaves due to leaf roll and that due to Fusarium wilt. In the latter case only the upper leaves are rolled at the beginning. In light cases or when the air is damp the wilt is often indistinct, but the diseased shoots die off before the healthy ones. In very serious cases or in very hot weather wilt of the infected sprouts is most pronounced. In the case of mycosis, in contrast with the non-parasitic leaf roll disease, single vines may become affected while others remain healthy.

An infallible proof of the presence or absence of the disease is furnished by a cross section of the stem. In diseased plants the vascular bundles are more or less brown, and microscopic examination shows the mycelium in the secondary pitted vessels. From the vessels of the stem, into which the fungus probably gained entrance through wounds, the fungus passes to the vessels of the stolons and tubers. Not all tubers of a diseased plant necessarily become infected. In the case of the tubers the fungus occurs first at the stem end. In spring the mycelium extends to the eyes and later enters the vessels of the young shoots. In Germany similar diseases occur, but so far it has not been certainly determined what species of Fusarium cause them. *Verticillium albo-atrum* causes a similar disease. It is possible that the roll disease even in the United States may not be due exclusively to *Fusarium oxysporum*, and I also suspect that other organisms may produce symptoms similar to those of the wilt disease.

For the United States vascular mycosis has a peculiar significance in that it seems to be especially prevalent in irrigated districts. In Germany I have observed that it is much more prevalent in dry years and dry localities than in years and regions well supplied with moisture, and from this I conclude that its prevalence in irrigated localities is intimately associated with the irrigation. A study of the best methods of supplying water, it seems to me, therefore, is not only of general interest, but is also of vast importance.

As in the case of fungi, bacteria can also invade the vessels and cause the disease which has been described as bacterial ring disease, and in which a rolling of the leaves may sometimes occur.

Whether such a vascular disease is produced by a single bacterial species has not yet been established. Similar diseases have been described by various workers. One especially worthy of mention is a disease recently described by Spieckermann and Kotthoff.[5] They have isolated a species named *Bacterium sepedonicum*, which grows very slowly, but on the other hand retains its pathogenicity in pure cultures for a long time. This disease is characterized by the spiral vessels being filled with a great number of bacteria while the pitted vessels are free or very slightly infected, and also by the large root vessels being for the most part free from bacteria and consequently interference with the water supply is not so great as in the case of mycosis. Gradually the enzymes secreted by the bacteria dissolve the walls of the spiral vessels and disorganize the surrounding parenchyma, and this results in softened places, which later give rise to cavities.

As the bacteria penetrate the tuber vessels from the vessels of the stem the disease is transmitted by the seed potatoes. In severe cases no shoots are produced, and as a result hills are missing or only sickly shoots, with longitudinally rolled leaves, develop. In less severe cases plants which appear normal are produced, but in the autumn these are paler and show a slight rolling of the leaves.

The disease is also influenced by the weather. A long period of drought results in a slow growth of the vines, the rolling appears earlier and is more noticeable, and the leaves become dry, beginning at the margin. This drying distinguishes the bacteriosis from the non-parasitic leaf roll disease, in which latter it does not occur.

[5] Spieckermann, A. Zur Kentniss der in Deutschland amphfenden Gefässkrankheiten der Kartoffelpflanze. Illustrierte Landwirthschaftliche Zeitung, **33**: 680–682. 1913.

——— Untersuchungen über die Kartoffelpflanze und ihre Krankheiten. 1. Die Bakterienringfäule der Kartoffelpflanze; In Gemeinschaft mit P. Kotthoff. Landwirtschaftliche Jahrbüche, **63**: 659–732. 1914.

The signs of the disease are not so pronounced in long-continued wet weather. In slightly diseased plants they are seldom or not at all noticeable. The dying of the vines is an indication of the disease. As ordinarily the different shoots of the same plant are attacked in varying degrees of severity they do not all die at once. An examination of such suspicious vines is the only means of determining with certainty whether the plant is diseased.

Seed potatoes in many cases show dark spots in the vascular ring. Sometimes, however, the diseased tissues are not at all or only slightly discolored, but the softening of the diseased area is characteristic.

Another group of diseases characterized by leaf roll is that of the so-called "foot-disease." I separate these also into mycoses and bacterioses. The prototype of the former is the Rhizoctonia disease and that of the latter the black-leg disease. Both mycoses and bacterioses attack the subterranean parts of the stem, which prevents an adequate transfer of water and nutrients and finally causes the death of the aerial portions. In mycoses this process is relatively slow, but in bacterioses it is comparatively rapid.

Rhizoctonia solani, regarding which we have the investigations of Rolfs and Morse, often attacks the potato seedlings and frequently destroys such large areas of tissue that the entire seedling, or at least its upper portion, dies. In the latter case side shoots develop, and even in older plants the early attack is indicated by the peculiar method of underground branching. Frequently infected vines also remain shorter, the internodes of the terminal portion of the axis being much shortened. The upper leaves are more or less rolled, and frequently more or less red or yellow, but it is seldom that all the leaves show rolling. The disease converts the lower part of the stem into a punky, brittle mass, and as the trouble progresses the tissues die and the shoot withers.

In the case of the foot diseases, infection of the tubers does not as a rule take place, but the mycelium and sclerotia of the fungus are frequently found on the surface of the tubers. I do not believe the killing of the fungus on the surface of the tubers a very promising means of controlling the disease. The mycelium and sclerotia of the fungus may indeed be killed by means of a solution of mercury bichloride, but the fungus is so generally distributed in the soil that this treatment by no means protects the potato from the disease. It seems to me, therefore, that it is necessary to investigate the biology of the fungus and to study more carefully all its relations to the soil.

In Germany I once observed a severe attack of Rhizoctonia on new unbroken forest soil. As the soil had a strong acid reaction I attempted to suppress the fungus by treating the soil with lime, and in this I was en-

tirely successful. It seems, however, that the acid is not the factor favorable to the development of the fungus, for in artificial media the parasite grows better on alkaline than on acid media. I suspect that the large air content of the freshly broken soil was the real cause of the unusually good development of the fungus in this case. Similar conditions also exist in Maine and in the Stockton delta of California, but in California it is not on the forest but on the peat soil, which is also porous and well aerated, that Rhizoctonia appears. That the fungus requires a large amount of air is evidenced by the fact that in artificial media it grows only at the surface, and also by the fact that the cultures when flooded with hydrogen peroxide rapidly absorb oxygen. Furthermore the fact that it actively attacks cellulose adapts it for growth on forest and peat soils. On filter paper moistened with a nutrient solution it grows so actively that in a short time the paper disappears and the culture loses over 80 per cent in dry weight. These latter facts require further elaboration, but they are offered here in support of my assumption. It is still an open question whether all foot mycoses are caused by Rhizoctonia. Notwithstanding the abundance of this fungus it is not improbable that fungi other than Rhizoctonia may attack the potato plant in a similar manner.

Bacteria, like fungi, may cause foot disease, as they also live in the soil and enter the stem through wounds. While fungi can penetrate the cell wall, bacteria are able to dissolve the middle lamella, which causes the stems to become soft and in many cases black. The whole process of destruction takes place so quickly that the stems very often become yellow and die within a few weeks.

In the beginning of the foot disease the leaves show a rolling, which is easily confused with that caused by other diseases, but the disease can be readily identified beyond question by the presence of the softened part of the stem, which does not occur in other troubles.

Several bacteria have been found to cause the disease—*Bacterium phytophthorum*, *Bact. atrosepticum*, *Bact. solanisaprum*, and *Bact. xanthochlorum*. It is remarkable that in artificial cultures each of these four species forms long threads in addition to the typical rod-like forms. Perhaps other bacteria may be found to cause black-leg. There are bacterial diseases which soften the stem without blackening it.

During prolonged spells of damp weather the tubers also are attacked in the soil. The bacteria enter the potato through the tiny wounds which are always present and produce a wet rot. This changes the inner part of the tuber into a kind of soft pulp. In such cases, however, the starch is not dissolved, and the cells lie separated.

In Germany, where it is often necessary to harvest potatoes in damp weather the bacteria not infrequently spread through the storerooms and

cellars and destroy a great many tubers. In America the danger from this source is small because of the dry autumns. Indeed it seems to me that black-leg of the potato is of but minor importance in the United States. On my trip I found it only in the far Northeast. I am told that in the Southern and Central States the infected tubers produce diseased plants, but that the disease does not live over winter in the soil, the apparent reason being that the bacteria do not form spores and are very susceptible to dry conditions. In my own experience I have found that a slight drying out is sufficient to kill small infections. The potato closes its wounds with cork and this enables the tuber to overcome these small infections. The same reason is responsible for the excellent results we have obtained in the drying off of potatoes. The tubers are spread out for some days before they are planted and in this way serious damage is prevented.

Finally I wish to call attention to another disease, which I saw in America for the first time and which has not yet been described. In the case of this disease also the leaves are often rolled, black spots occur on different places on the stem, and a mycelium is found in the interior. While in the well-known vascular mycoses the mycelium grows in the cavities of the pitted vessels and but few threads are found, in this unknown disease the primary vessels (the ring and the spiral vessels) are filled with hyphae. Professor Stakman, of St. Paul, who is working on the disease, told me that the mycelium belongs to a Fusarium species. Some scientists think the disease a kind of black-leg which has assumed another form because of the plants growing under other conditions. In that case the mycelium would be of a secondary character. I can not share this opinion. I believe the disease is a particular form of vascular mycosis and that when bacteria occur in the vessels they are of a secondary nature. The problem, however, can be solved only by thorough investigation.

The following diagrammatic grouping briefly summarizes the diseases discussed:

I. *Crisp leaves:*
   1. Curly dwarf disease.

II. *Rolled leaves:*
   1. Non-parasitic—
      A. Leaf-roll disease.
   2. Parasitic—
      A. Vascular diseases:
         a. Fungi—Wilt disease.
         b. Bacteria—Ring disease.
      B. Foot diseases:
         a. Fungi—Rhizoctonia disease.
         b. Bacteria—Black-leg.

A diseased potato plant can not be cured, therefore our efforts should be directed toward prevention rather than cure. Diseases which are carried by the seed potatoes can be prevented. The vascular mycoses and bacterioses are found in the seed tubers when cutting them, the vessels being brown in case of the former and softened in the latter. The problem is somewhat more difficult in the case of black-leg. When the tubers show large areas of infection the disease may be recognized easily but small areas may escape attention. There is no way of determining, however, whether a potato is the product of a plant which was affected with curly dwarf or leaf roll. The farmer should know the disease in the field and mark the diseased vines so that the tubers may not be used for seed. Though not first class the quality of potatoes produced by such plants is relatively good for food so that it is not necessary to destroy the plants.

In Germany we have arrangements for field inspections so that the farmer may be sure of getting rid of these diseases. Whenever a farmer wants an inspection made he applies to the Deutsche Landwirtschafts-Gesellschaft (German Agricultural Society) in Berlin, or to a Landwirtschaftkammer (Board of Agriculture), one of which is in nearly every state or province of the Empire. The application must show the variety of potato, source of seed tubers, and the area to be inspected. When the potatoes are in bloom a Commission, consisting of an agriculturist and a pathologist, inspects the field. If 5 per cent of the plants are found to be diseased, the certificate is withheld and if a smaller per cent is found the grower is required to remove the affected plants. Later a second inspection is made and if diseased plants are found the certificate is refused. At this inspection samples are taken from several different parts of the field in order to determine whether the yield is uniform. An ideal seed potato should not only be healthy, but should come from a healthy and prolific mother plant. This is necessary to insure healthy progeny. The farmer is glad to bear the expense of the inspection, knowing he can sell his certified seed at a good price.

This system of inspection is going to be introduced in parts of the United States, that is, in the states having the most progressive potato culture, such as Maine, Wisconsin, etc., and I am sure it will be successful.

Finally, it may be said that the basis for all measures of control is a thorough knowledge of the diseases, and our endeavor should be to spread this knowledge as much as possible.

BERLIN-DAHLEM
GERMANY

THE LOOSE KERNEL SMUT OF SORGHUM[1]

ALDEN A. POTTER

WITH PLATE X AND TWO FIGURES IN THE TEXT

In a paper on the smuts of sorghum in America, the writer (1912)[2] recently noted the occurrence of *Sphacelotheca cruenta* (Kühn) Potter in this country. It has become evident on further investigation that it has been largely confused with the "closed" or "covered" species, *Sphacelotheca sorghi* (Lk.) Clint. The present paper is intended to clear up this confusion by adequate illustration and discussion of the taxonomy of the species, *Sphacelotheca cruenta*.

This smut was first described by Kühn (1872). Link's (1825: 86) rather obscure original description of the other species under the name *Sporisorium sorghi*, might perhaps seem more fitly to apply to this one because he notes ruptures in the substance of the seed and excrescences in the outer ends of the panicle branches. Kühn (1875) himself, however, examined Ehrenberg's specimens, from which Link described his species, and since he considered that the fungus should be transferred to the genus Ustilago in which Passerini had by this time placed a doubtful species named *U. sorghi*, he re-named it *Ustilago tulasnei*.[3] It is thus evident that he considered Link's species distinct from his own species, *Ustilago cruenta*, which he had previously described in 1872.

Kühn's descriptions of the latter[4] appear to be scarcely complete, however, and have apparently led to considerable confusion of the two kernel smuts in literature. The marked emphasis which he laid both upon the formation of sori in the stem (more particularly in the panicle branches) and upon the red coloration from which he drew the specific name, *cruenta*, seems to have been unfortunate, and later writers do not bear him out in this. Thus Brefeld (1883: 91) from material sent him by Kühn, gives the following description:

[1] The writer is greatly indebted to Dr. G. P. Clinton of the Connecticut Agricultural Experiment Station for advice and assistance through correspondence.

[2] Bibliographic citations in parentheses refer to "Literature cited," p. 154.

[3] This species, i.e., the covered kernel smut, having been transferred to the genus Sphacelotheca by Clinton (1904: 385), Kühn's objections to the specific name, *sorghi*, no longer obtain under the present rules of nomenclature.

[4] Besides the original description, cited above, see also Kühn, 1877: 81.

"Das *schwarze*[5] Sporenlager des Brandpilzes hatte einen eigenthümlichen Stich ins Rötkliche, die einzelnen kleinen rundlichen Sporen (von 6–12 μ Grösse) waren eher gelb als braun gefärbt, ..."
and again, (1895: 43):

"Es ist bekannt, dass der Hirsebrand, Ustilago cruenta *Kühn*, ... Hirseformen bewohnt und dass die befallenen Fruchtknoten in eine *schwarze*[5] Brandmasse umgewandelt werden ohnlich wie es bei dem Flugbrande des Hafers geschieht."

He further observes here, moreover, that the sori in a large number of infected plants of *Sorghum saccharatum* were confined to the inflorescence, and mostly to the ovaries, unlike Kühn's descriptions of the type.

Brefeld is not consistent in his classification. He at first (1895: 120) considered *U. tulasnei* and *U. sorghi* the same species. Later (1905: 57) he apparently held the same opinion regarding *U. sorghi* and *U. cruenta*, while his most recent publication (1912: 31) classes all three as distinct species, *U. tulasnei* being distinguished from the others by more sparse conidial formation and a tendency toward hyphal structure in the growth on artificial media. *U. cruenta* is separated only because of the formation of sori on the stem. Although the growth of the two American species has been found by the writer to differ at times on nutritive media, this seems so largely dependent on the character of the medium and other conditions of growth that the lack of a record of these conditions in Brefeld's work makes his classification on the basis of cultural studies seem of doubtful value. Clinton (1897: 375) has called in question the species dealt with by Brefeld in his "chief investigations," largely because of the close similarity between the spores and germination of the two species. These doubts seem dispelled, however, by the more thorough understanding of their real differences, now at hand.

While Brefeld's illustrations (1895: Pl. I, figs. 12 and 13), in connection with his statement that sori seldom appear on the branches (1895: 43) are fairly convincing as to the identity of the American species, it remained for Busse (1904: 373) to note the fragile character of the membrane on the sorus—its most distinctive, constant characteristic. He also calls attention to the fact that Kühn's material was probably not typical and states that, in his material, plants infected by this fungus did not commonly show the red coloration nor crumpling of panicle branches described by Kühn.

From these descriptions we are able to identify the American species here under discussion with Kühn's *Ustilago cruenta*. But for a certain variability which this species has shown in its development on different sorghum varieties in plantings made by the writer (these are described

[5] The italics are our own.

below), it might readily be suspected, with Clinton (1897: 375), that the later authors, Brefeld and Busse, were not dealing with the same species that Kühn had. Busse (1904: 371) has called attention to the possibility of this variation, dependent upon host varieties, in describing the other species of kernel smut, *Sphacelotheca (Ustilago) sorghi*.

Using spores from a single lot the writer made inoculations at Amarillo, Texas, in 1911, on six different varieties of sorghum. Milo proved immune, as it has from the other smut forms. A saccharine sorghum, the Red Amber variety, yielded 10.9 per cent of infected plants, and like Brefeld's descriptions, the sori were for the most part confined to the ovaries. This was true also of kafir (Pl. X, fig. 1), of which 13.8 per cent was smutted, and broomcorn with 6.9 per cent infection.

On the other hand a Brown kaoliang, showing 10.3 per cent smutted plants, and Freed sorgo, a sub-saccharine form somewhat similar to the kaoliangs (with 5 per cent infection), frequently showed the typical lesions described by Kühn. Two panicles affected in this way are shown in Plate X, figures 3 and 4. These lesions, like all injuries on sorghum —particularly in the case of saccharine varieties, developed more or less of a reddish color. This, however, is plainly a characteristic, not of the parasite, but of the host. Even in the saccharine varieties the coloration is at least not conspicuous in sori which happen to be confined to the flowering parts.

In Plate X, figure 2, is shown a young smutted inflorescence of Freed sorgo preserved with many unbroken sori, much as in one of Brefeld's illustrations (1895: Pl. I, fig. 12). This is unusual, particularly in the Great Plains areas where the high winds break the thin membrane as soon as it emerges from the sheath. The most typical form is shown in figure 1 of the plate. The long, naked columellae may be seen projecting from the glumes, the whole head appearing, as Brefeld aptly expresses it, much like a chimney-sweep's broom.

Parallel inoculations with *S. sorghi* made at the same time, and on the same varieties as those with *S. cruenta* showed the two forms to be entirely distinct macroscopically. This will appear from a previous illustration by the writer (1914: Pl. XXXII, fig. 1).

To these macroscopic distinctions should be added a few statements as to microscopic characters. The spores of the two kernel smuts are difficult to distinguish. Those of *S. cruenta* are somewhat more irregular in size and shape and thus average a very little larger. In measurements made by the writer their average diameter has usually been slightly more than 6 μ, while the other species has averaged slightly less, although this is not always true. Dr. G. P. Clinton has found, however, in examining our own and other exsiccati, that the fragility of the membrane in the

loose kernel smut is apparently due not only to its thinness (fig. 1) but also to the fact that the "cells are more nearly spherical and separate easily into small groups or individual cells which are usually much larger than the spores: while the membrane of *S. sorghi* is more permanent, less easily broken up, and consists chiefly of cells that are more elongated, with shorter diameter, usually not larger than that of the spores, and when pressed apart are apt to adhere in thread-like groups."[6] These large, spherical, hyaline cells (fig. 2 *a*) can be found characteristically scattered

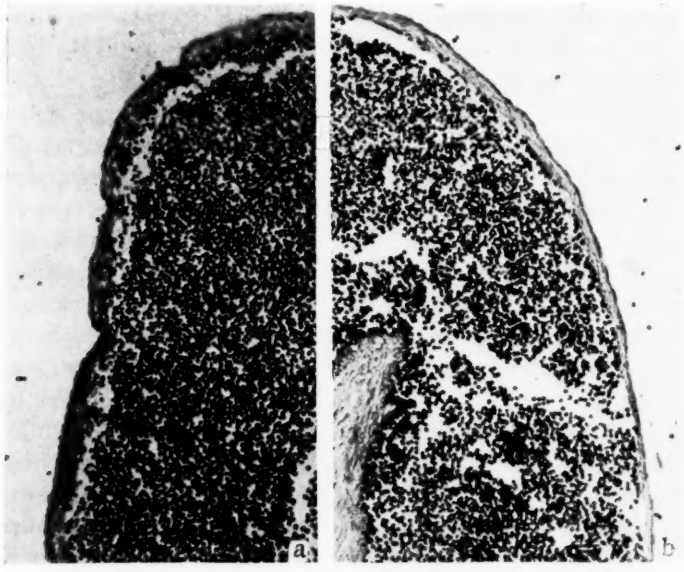

FIG. 1. *a*, sector of a longitudinal section through apical portion of a young sorus of Sphacelotheca sorghi on kafir, grown at Amarillo, Texas, in 1912. Enlarged 100 diameters; *b*, preparation similar to that of *a*, but of S. cruenta. A portion of the columella appears in the figure. Note also the membrane, which is typically much thinner than in the other species. Photomicrographs made by the author, 1914.

through the escaped spore mass in herbarium material. They usually measure up to 12 μ or more in diameter, although some may be no larger than the spores.

American literature has not recognized this species although it has apparently been present for many years. Specimens collected by officials of the Office of Cereal Investigations of the United States Department of Agriculture show it to have been in this country previous to 1906. It also appears that the oldest specimen of sorghum smut in American exsiccati, Ellis' North American Fungi, No. 1496, collected by Trelease in

[6] Letter of March 30, 1914.

Wisconsin, is not of *Sphaecelotheca sorghi*, as reported, but of *S. cruenta*. Trelease (1885: 138) apparently noted its lack of the typical appearance but did not consider it distinct. His observation that it was imported with Chinese seed agrees with its appearance among the kaoliangs in the plantings of the Office of Cereal Investigations. Dr. G. P. Clinton has also kindly submitted a specimen collected by Thaxter from Kingston, Jamaica in 1891. The only other specimen in American herbaria, so far as is known to the writer, is one in the Farlow herbarium collected by Kühn, himself.

That the species has also been confused in European herbaria would appear from an examination of such specimens as Jaczewski's Fungi Ros-

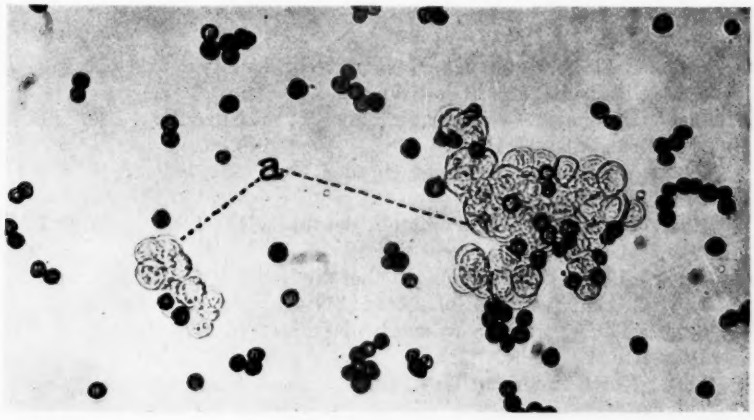

FIG. 2. Typical spore material of Sphacelotheca cruenta. × 350. Note the large, sterile cells in two groups at *a*. Photomicrograph made by the author, 1914.

siae Exsiccati, No. 206, collected in Manchuria in 1896, and Vestergren's Micromycetes Rariores Selecti, No. 1209.

A brief study of the amenability of this smut to control by seed treatment was made in 1912. The formalin treatment (Freeman and Umberger 1910: 6) was found to eliminate infections of 12.5 and 15.7 per cent in kafir and broomcorn, respectively. It should be said that in these tests, and in others with *S. sorghi* on different varieties, it has been found that seed which has retained the glumes through the thrashing process, as does broomcorn, will usually sustain, without injury, a more severe treatment than naked seed, and at the same time requires a more severe treatment in order to be certain of prevention. This protective action of the glumes appears again in the fact that usually more severe infection is sustained in the field by plants grown from smut-infested seed which has no adhering glumes.

SUMMARY

The confusion of two, distinct, American species of kernel smut on sorghum has resulted largely from the undue emphasis laid by Kühn on certain peculiarities of the species, *Ustilago cruenta*, which he described in 1872.

The occurrence of this species in America as early as 1885 is established. It is shown that its most distinctive character is the fragility of the membrane enclosing the sorus. This is dependent not only upon its thinness, but also upon the more nearly spherical shape of its cells.

Infection may be prevented by the usual seed-disinfecting treatments.

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POTTER: LOOSE KERNEL SMUT OF SORGHUM

EXPLANATION OF PLATE X

Sphacelotheca cruenta on *1*, kafir, *2*, Freed sorgo, *3*, Brown kaoliang, and *4*, Freed sorgo. Enlarged. Note that in *3* and *4* the stem and branches of the panicle are involved. In *2* the sori are comparatively intact because the specimen was taken when young, while in *1* they are all broken and most of the spores dissipated. Photographed by Mr. J. F. Brewer, 1914.

SOME NEGLECTED PHASES OF PHYTOPATHOLOGY

J. G. GROSSENBACHER

INTRODUCTION

The influence of the environment on the development and maturation of tissues is evidently far-reaching, but scarcely a beginning has yet been made in determining it. The effects of sub-optimal conditions on growing plants are largely unknown except in their extremes and only very imperfectly even there. Phytopathology has been too much concerned with the morphology and physiology of the microorganisms associated more or less constantly with many diseases of cultivated plants, to permit continuous and thoroughgoing studies of the seasonal and life history of the tissues first affected by the early stages of disease. This has tended to discourage investigations into the conditions that precede such visible derangements in plants. In fact, this one-sided development of phytopathology has been so marked in the United States during the past fifteen years that the special departments devoted to this work are still concentrating most of their teaching energies on the study of fungi and bacteria along with methods of preventing the diseases with which some of these organisms are connected. The early development of human pathology was not thus sidetracked because in those days the relation of certain microorganisms to disease had not yet been discovered. By the time Pasteur appeared upon the scene medical schools had developed so far along histological and physiological lines that the germ theory of disease only added to and modified their courses but did not eliminate or wholly supplant further research along the old lines. Phytopathology has not this foundation. General physiology and histology along with Küster's Pathologische Pflanzenanatomie would furnish most of the general principles on which such a foundation could be built. At the present time these more basic considerations are merely tolerated and not amalgamated with what passes for plant pathology. The prime importance of the physiology and histology of the host as affected by the environment is usually not even recognized. A man proficient in the knowledge available on the pathogenic microorganisms related to the diseases of human beings would not be granted permission to practise medicine, while one so prepared in regard to some of the organisms associated with most of the best known diseases of plants is given high rank in plant pathology.

However, plant pathology has attained some results of most extraordinary economic importance in the United States in recent years, especially on the prevention of certain diseases of herbaceous plants, and on some of those affecting the leaves and fruits of woody plants. On the other hand, very little actual progress has been made in our knowledge pertaining to the diseases affecting the bark of woody plants and those of the phloem of herbs; that is, of the diseases which originate in the interior, or to those which are related to the development and life history of the more permanent tissues. Practically the same is true as regards a real understanding of the primary causes of fruit-rots. We know, for instance, that careful handling and provision for good aeration in getting fruits to market usually reduces the amount of rot decidedly, also that in case of certain fruits spraying with fungicides is of great value; yet during years of certain peculiar weather combinations when fruits drop easily or decay even on trees low temperature must be used to prevent rot in marketing. In such seasons our ignorance of the real combination of factors leading to decay is brought forcibly to our attention and most of us are willing to shift the scene.

European phytopathologists have been following the lead of those in the United States in the more practical phases of the work. The growers in Europe are not in as close touch with their investigators, and have not compelled as much attention. The development of the spraying business has therefore lagged behind that in this country. Nevertheless their advances have been notable and perhaps more fundamental than ours. They have been investigating many of their problems more from a standpoint of the host and its environment. Some recent contributions from there show that such considerations are assuming more and more prominence in their research.

SORAUER'S WORK

This trend in Europe is also brought out by comparing the first, second and third editions of Sorauer's Handbuch der Pflanzenkrankheiten. That veteran pioneer in plant pathology has developed some most promising points of view and methods of attack, many of which are so far ahead of his time that they are attracting but minor attention and therefore enlist few followers. He has dared attack the more abstruse problems. His extensive studies on plant diseases during the many years that he has given to the subject show a gradual change of view in regard to the primary causes of diseases of certain types. This is especially indicated by comparing the space devoted to diseases of parasitic and those of non-parasitic origin in the three editions of his Handbuch. In the first edition 147 pages are given to those of non-parasitic and 190 pages to those of

parasitic origin. In the second edition non-parasitic diseases are discussed on 715 pages and those due to plant parasites on 430 pages, while to insect and other enemies 116 pages are given. In the third edition non-parasitic diseases occupy 877 pages, those due mainly or in part to plant parasites 535 pages, and troubles chiefly due to insects and other animals take up 747 pages.

Although one may not always agree with the interpretation of the facts about diseases described in the first volume of the last edition, the mass of material collected and classified in its pages is invaluable as a source of reference as well as of inspiration. His discussions of the environmental factors as related to the development of specific diseases, and the histological details given about many of them are illuminating and suggestive in most cases.

LEAF-ROLL DISEASE OF POTATO

A problem that affords a fair illustration of the more obdurate diseases to understand is leaf-roll of potato. The various attempts that have been made in the past to fathom the cause either failed or at least were only partial successes. The present status of the problem, however, is much better than that of many others which have long since been dismissed from further investigation as solved. In this case one can readily imagine he discerns something not far removed from the first marked effect of the cause, even though it is seen only with a telescope instead of with a microscope; for that reason one can as yet give only certain characteristics of it.

In a recent paper on this disease of potato Quanjer[1] not only discloses some very important facts regarding this particular malady but makes use of a method of approach that should be more general in the study of diseased plants. He found that the disease in question is not due to pathogenic organisms but that it is connected with a necrosis of the phloem strands. As is well known, affected plants ordinarily remain small, have short stolons, their leaves are more or less rolled up and the yield of tubers is much reduced. In the plants affected by leaf-roll both the extra- and intraxylar strands of phloem were always found discolored and collapsed more or less. In milder cases only the oldest phloem strands or those nearest the bast-fiber groups inside as well as outside of the xylem, were involved. A disorganization of the phloem is said to take place which is apparently accompanied or preceded by a slight lignification of

[1] Quanjer, H. M. Die Nekrose des Phloems der Kartoffelpflanze, die Ursache der Blattrollkrankheit. Meded. Rijks Hoogre Land, Tuin en Boschowsch. (Wageningen) **6**: 41–80. 1913.

the cell walls. Measurements made on the phloem cells of normal and affected strands of three varieties of potatoes showed the diseased cells to be on an average from 26 to 36 per cent shorter than the normal ones. It is thought that the shrinkage and lignification of the affected phloem exert a pull or tension on the surrounding tissues, owing to the fact that the parenchyma cells adjoining the affected phloem are found elongated or drawn-out toward the collapsed phloem.

The first traces of the discoloration of the phloem strands were sometimes found present in young stems after a few leaves had become unfolded, and the discoloration is said to follow elongation growth upward and at such a rate that the phloem of the new internodes shows indications of it before that portion of the stem has attained full size; in other cases the discoloration did not become evident until the plants were large, or after the "seed" tuber had disintegrated. In 1912 some of the early stages of the disease were found in the distal internodes of plants as late as August and September. The degree or extent of injury was found to decrease toward the distal ends of the plants as well as toward the "seed" tuber underground, although the affected strands may be traced to both leaves and "seed" tubers. In the lower aerial internodes where considerable secondary growth occurs, the injury is also chiefly confined to the oldest extraxylem and to the intraxylem strands of phloem.

The black discoloration of the dead spots in the leaves of such affected plants is thought to be due to an injurious accumulation of proteins as well as that of mineral substances in them owing to the stoppage in the downward flow of proteins through the phloem. It is noted that the varieties of potato which are ordinarily resistant to Phytophthora blight are severely blighted when affected by phloem necrosis. Plants dying from phloem necrosis do not become defoliated when they die but the leaves, surcharged with elaborated food, die and turn black and remain attached to the stems. Quanjer thinks that lignification and other processes are instituted during the dying of the phloem cells, though this was not definitely established. The chief difference between the normal change of the older phloem to its functionless and collapsed state as it occurs in the bark of trees, et cetera, and that occurring in the necrotic collapse of the phloem in such diseased potato plants is thought to consist in the biotic changes, which are absent from normally dying phloem.

The lignification of the phloem has been noted in a number of apparently normal plants although the question has been studied but little. Vesque[2] found that in *Betula alba* lignification of the phloem may occur

[2] Vesque, J. Memoire sur l'anatomie comparée de l'écorce. Ann. Sc. Nat. Bot. Ser. 6, **2**: 82–198. 1875. (178–79).

comparatively early in the life history of the tissues. Schumann[3] also noted the occurrence of lignified phloem cells in *Scorzonera hispanica* and *Aster thyrsiflorus*. The sieve plates were often found especially strongly lignified in these plants. Boodle[4] has more recently studied some plants showing more or less lignification of the phloem. In the thicker portions of sunflower stems he found that in August patches of phloem-ray cells and the walls of some sieve tubes were lignified, while in October lignification of both the sieve tubes and companion cells had occurred and the cell contents showed the lignin reaction. Sometimes only scattered groups of cells in the phloem bundle possessed lignified walls. Boodle thought that cultural or environmental conditions and the relative robustness of plants determine the degree of lignification that will occur in the phloem.

Quanjer thinks that phloem necrosis of the potato is due to a slight functional derangement in the phloem which interferes with the normal transportation of elaborated foods and results in its accumulation and thereby giving rise to some poison which induces the disease. Susceptibility to this malady is thought to be inherited. He notes that in cases where discoloration or phloem injury is slight, lignification is correspondingly slight, and for that reason assumes that the dying process induces lignification.

It is a well-known fact that frequently after the protoxylem cells and strands of the stems of various plants are practically mature and more or less lignified they are stretched and even ruptured by the further elongation of the axis. In many plants such stretched and ruptured protoxylem cells occur in nearly all parts of the axis, but the phenomenon is especially common in the basal portion of stems and branches where the elongation and maturation processes more often interlock. Since the measurements of diseased phloem cells made by Quanjer show that they are from 26 to 36 per cent shorter than the normal ones of the same stem, and since such affected cells are in the older portions of the phloem it appears at least possible and perhaps probable that the causes of injury are very similar to those inducing the injury and rupture of protoxylem cells. The normally short-lived nature of protoxylem and its small content of protoplasm in connection with the fact that its function is so quickly supplanted by that of the regular xylem are doubtless the chief reasons why no serious disturbances follow such protoxylem injuries. The disintegra-

[3] Schumann. Beitrag zur Anatomie des Compositenstengels. Bot. Centbl. **41**: 193–96. 1890.

[4] Boodle, L. A. On lignification in the phloem of Helianthus annuus. Ann. Bot. **16**: 180–83. 1902.

tion of the protoplasm of injured phloem cells not only interferes with the normal transfer of elaborated foods but seems to give rise to some diffusible substance that may injure and kill adjoining cells. However, this does not explain how it is that in a growing stem lignification may overtake elongation growth and thus result in injuries, yet it suggests that such things might happen if the environment were unfavorable to elongation growth during the early developmental period of an axis.

I consider this work of Quanjer of much importance because it is a type of research in phytopathology that is needed in a number of our most dreaded plant diseases, including some of both woody and herbaceous plants. For example, the bark diseases of trees and shrubs and some of the other stem diseases of herbaceous plants could doubtless be studied with profit from the standpoint of the relation between the life history of the affected tissues to the development of such diseases as was done by Quanjer.

BARK DISEASES OF FRUIT TREES

During the past five years I have been studying certain bark diseases of fruit trees. Most of the first three was devoted to a trouble of apple trees that results in the death and decay of the bark on the trunk at the ground, and is known as crown-rot.[5] Since then much time has been given to study of a very similar disease of Citrus that is usually known as foot-rot. The similarity of these two troubles consists chiefly in the fact that they begin with the disintegration of the inner phloem or cambium.

Crown-rot was concluded to be of nonparasitic origin. It was found most prevalent following winters in which the first cold period came while the bark at the base of some tree trunks was still growing. Similar injuries developed at crotches and other places where elaborated food is most abundant in fall. A fairly complete series of the various stages in the development of so-called cankers and crown-rot were secured by fixing specimens collected from late winter to mid summer, in killing fluid.

The simple statement that there is a connection between the initial stages of this trouble and the occurrence of low temperature affords no explanation of the cause. For that reason most of the available literature on tree growth and its relation to the factors of the environment, was collected and reviewed with the hope of gaining some information and points of view that might prove of use in the study of crown-rot. The result was gratifying in that a great and varied fund of ideas was

[5] N. Y. State Agr. Exp. Sta. Tech. Bulls. 12 (1910) and 23 (1912).

secured,[6] but proved rather disappointing in that only occasional suggestions having direct bearing on the problem in hand, were found. My observations which showed that late radial growth may be very irregularly distributed were found to have been made also by some other investigators. This review of the literature together with my own studies suggest a few fairly plausible ideas on some of the causes of crown-rot. These notions are not established facts, however, and whatever value they may have lies mainly in pointing the need for further work. Most of my attention was necessarily given to the development of the disease from the initial injuries by making a histological study of the material collected preceding and during growing seasons.[7]

A similar plan of attack has been followed while studying foot-rot and gummosis of Citrus during the past two and a half years. The strange and perhaps unexpected likeness of the irregular distribution of late-season radial growth was also brought to light in regard to Citrus. It was found, too, that cold snaps induce injuries in some such actively growing regions of bark. It appears probable, however, that at least in Florida some other adverse factors of the environment, may also induce phloem and cambium injuries that subsequently result in bark diseases. The material for a histological study of the development of these Citrus troubles does not make a complete series as yet, but enough is at hand to permit the publication of a preliminary report on the subject once the final paper on the crown-rot of apples has been finished.

This rambling discussion of what are deemed types of phloem and bark diseases of herbs and woody plants does not aim to present details but only to call attention to some of the problems in phytopathology that deserve more general attention not only because they are of fundamental scientific interest but also because they are equally important from an economic standpoint. Such diseases not only damage or destroy one crop but actually kill trees which it has taken many years to produce.

The study of problems of this kind does not yield the self-satisfying and quick results sought by most investigators. To spend a few months in trying to determine the cause of such a malady as leaf-roll, fruit-rots, or any of the various bark diseases of trees like peach yellows, for instance, and then spend a half dozen years in studying the morphology, physiology and life history of any microorganisms that may be found trespassing on such affected plants yields only minor advances and diverts attention

[6] The periodicity and distribution of radial growth in trees and their relation to the development of annual rings. Trans. Wisc. Acad. Sc. Arts and Letters **18:** — 1915.

[7] A paper is now in preparation on this phase of the subject.

from the fundamental problem to be solved. It is more conducive to progress to become acquainted with only a portion of a main line than to run to the end of a short branch, because other investigators may explore different sections of the field and thus enable someone to bridge the remaining gaps and give us a complete solution of the causes. Once the causes of a disease are fully known preventives or correctives can usually be found with much less labor.

U. S. DEPARTMENT OF AGRICULTURE
WASHINGTON, D. C.

EXPERIMENTS ON THE SUSCEPTIBILITY OF SWEET POTATO VARIETIES TO STEM ROT

L. L. Harter and Ethel C. Field (Tillotson)

The large losses sustained by sweet potato growers from the stem rot in New Jersey led the writers to make a study of this disease in the hope of discovering some remedial measures. The results of this investigation, which extended over two years, were published in Phytopathology[1] (1914), where the symptoms of the disease, the causal organisms (*Fusarium hyperoxysporum* Wr. and *F. batatatis* Wr.), extensive inoculation experiments and various other features were discussed. In all the experiments the Yellow Jersey variety, which is grown extensively in New Jersey, Delaware, part of Maryland and a part of Virginia, was used. A study of a disease of a single variety, however, in a limited region can not be taken as typical of the crop in general, especially in a country where so many different varieties of sweet potatoes are grown under such diversified climatic conditions. It was well known that stem rot did not occur in some localities, but it was not certain whether this was due to the fact that the disease had not been introduced or whether resistant varieties were being grown. Investigation indicates that both suppositions are true. In fact, regions are known where, at the present time, a very susceptible variety is growing entirely free of stem rot, viz: Nancy Hall at Newton, North Carolina and at Orangeburg, South Carolina. Furthermore, certain varieties are known to be grown entirely free from stem rot in localities where a large percentage of susceptible varieties are affected by the disease, viz: Yellow Yam at Lincoln, Arkansas and Triumph at Foley, Alabama.

Since the disease is a vascular trouble and is propagated largely through infection of the plants in the field, there seems to be but two methods through which control may be sought; first, that of obtaining a variety or varieties resistant to stem rot, or, secondly, of obtaining a strain in a variety which is resistant. The present paper gives the results of variety tests which were carried on during the two seasons of 1913 and 1914.

Experiments were conducted at Vineland, New Jersey, on the Potomac flats, Washington, D. C., and at the Arlington Experiment Farm. In

[1] The stem rot of the sweet potato, Ipomoea batatas. Phytopathology **4**: 279–304, plates 24-26. 1914.

1913 at Vineland, seven varieties obtained from the Office of Horticultural and Pomological Investigations were planted in infected soil. These same varieties and others were planted on the Potomac flats in 1913 and at the Arlington Experiment Farm in 1914 and inoculated with *Fusarium hyperoxysporum*. All inoculations were made by inserting spores and hyphae into wounds made at the base of the stems, care being taken not to injure the plants any more than necessary. *Fusarium hyperoxysporum* was used for all inoculations because it is the more vigorous parasite of the two species causing stem rot and, likewise, the more widely distributed.[2] The cultures used were in the stage known as high culture, that is, a culture containing a maximum of septate, sickle-shaped spores and a minimum of hyphae. No attempt has been made to test out all varieties of sweet potatoes. There are more than 200 commercial names for this crop, but probably less than 30 true varieties. Most of those of commercial importance have been included in these tests.

The results of the experiments are recorded in the following tables:

DISCUSSION OF RESULTS

The first records of the results of inoculations were made merely from a superficial examination of the plants. It is not always possible to tell definitely whether a plant is infected without opening the epidermis. This was not done, however, until later in the summer, since very young plants might be killed by such mutilation rather than by the fungus. Later in the season when the plants were strong, the epidermis was frequently opened whenever there was any doubt.

It is seen from table 1 that 15 different varieties were inoculated in 1913. Successful infections were produced on plants of eight of these varieties. All plants were recorded as infected when the bundles were blackened for a few inches in each direction from the point of inoculation. It must be remembered that by the use of such a method many plants were recorded as infected which were not materially injured. In most cases those varieties which showed the largest percentage of infection were the ones most severely injured by the fungus, as, for example, in table 1, Early Carolina, Nancy Hall, Red Jersey and Yellow Jersey. It should be noted also that the fungus invaded the roots of some of the plants of these varieties. The organism was recovered from the vines and roots of many infected plants.

In 1914, inoculations were made on 12 of the varieties used in 1913 (table 3). The Yellow Jersey, Early Carolina and Red Bermuda were

[2] Stem rot has been found to occur in the states of New Jersey, Delaware, Maryland, Virginia, Ohio, Illinois, Missouri, Iowa, Kansas, Oklahoma, Arkansas, North Carolina, Georgia, Alabama and Mississippi.

omitted. Plants of the Red Bermuda could not be obtained and the susceptibility of Yellow Jersey and Early Carolina to stem rot was so well known that further experiments were thought unnecessary. Four varieties, the Yellow Yam, Miles Yam, Vineless Yam and General Grant Vineless Yam, were used in 1914 but not in 1913. It will be seen from table 3 that some plants in all varieties were infected, the number in several cases, however, being quite small. As in the previous year some of the plants recorded as infected were not very greatly injured. Roots from one plant each of five varieties, viz., Georgia, Vineless Yam, Yellow Yam, Key West Yam and Nancy Hall, became diseased.

From table 2 it is seen that some plants of all varieties except Southern Queen were infected when planted on infected soil, the percentage in some cases being high. The roots of a number of plants were likewise diseased.

These experiments show that the varieties Early Carolina, Yellow Jersey, Red Jersey and Nancy Hall are particularly susceptible to stem rot under natural conditions in New Jersey and when artificially infected at Washington, D. C.

These same varieties are equally susceptible to the disease when grown normally under field conditions in other sections of the country. The Big Stem Jersey and Yellow Jersey varieties are grown extensively in Illinois, Iowa and Kansas and in these sections suffer as much from stem rot as they do in New Jersey. Nancy Hall, a variety not grown in New Jersey or Delaware on a commercial scale, appears to yield to the stem rot as readily there as in Iowa, Arkansas, North Carolina or Alabama and at Washington, D. C., when artificially infected. On the other hand such varieties as Dahoney, Red Brazilian and Yellow Strassburg which show a high degree of resistance when grown normally under field conditions were likewise the more resistant when artificially inoculated. In general these results seem to indicate that inoculation experiments furnish a fair standard for judging the relative susceptibility of sweet potato varieties to stem rot. Two other varieties,[3] Porto Rico and Providence, on which no inoculations were made, are quite susceptible to stem rot under field conditions in Alabama.

However, the substitution of one variety of sweet potatoes for another in any region cannot be done alone upon the basis of their resistance to disease. In the first place, it is a well-known fact that a variety which is prolific in one region is not necessarily so in another. For instance, the Yellow Jersey, which produces a potato of exceptional quality and shape in New Jersey and Delaware, produces a very stringy, rooty potato in the South. Furthermore, some of the very productive sweet potatoes

[3] Harter, L. L. Notes on the distribution and prevalence of three important sweet potato diseases. Phytopathology **5**: 124–126. 1915.

TABLE 1

Results of inoculating different varieties of sweet potatoes on the Potomac Flats, June 15, 1913

VARIETY	NUMBER INOCULATED[1]	NUMBER INFECTED JULY 3	NUMBER INFECTED JULY 16	NUMBER INFECTED OCTOBER 14[2]	ROOTS INFECTED	PER CENT OF INFECTION
Nancy Hall	34	27	27	28	4	82.3
Early Carolina	37	16	19	22	1	59.4
Yellow Jersey	35	0	10	16	4	45.7
Red Jersey	40	10	16	18	3	45.0
Georgia	35	6	8	8	0	22.8
Southern Queen	68	9	9	9	0	13.2
Eclipse Sugar Yam	32	4	4	4	0	12.5
White Yam	33	2	2	2	0	6.0
Florida Yam	39	0	0	0	0	0.0
Pierson	38	0	0	0	0	0.0
Yellow Strassburg	43	0	0	0	0	0.0
Key West Yam	32	0	0	0	0	0.0
Red Bermuda	40	0	0	0	0	0.0
Red Brazilian	40	0	0	0	0	0.0
Dahoney	40	0	0	0	0	0.0

[1] Ten checks were used for each variety. These plants were punctured at the base of the stem in the same way as those inoculated. None became infected.

[2] Potatoes were dug and final records made on October 14.

TABLE 2

Results of planting different varieties of sweet potatoes on infected soil during season of 1913 at Vineland, New Jersey

VARIETY	NUMBER PLANTED	NUMBER INFECTED JULY 15	NUMBER INFECTED AUGUST 12	NUMBER INFECTED OCTOBER 15[1]	ROOTS INFECTED	PER CENT OF INFECTION
Early Carolina	110	0	9	28	8	25.0
Yellow Jersey	282	7	17	54	12	19.1
Red Jersey	105	1	3	14	5	13.3
Nancy Hall	110	0	0	6	0	5.4
Georgia	76	0	0	2	0	2.6
Miles Yam	109	0	0	1	0	0.9
Southern Queen	109	0	0	0	0	0.0

[1] Potatoes were dug October 15 and a record made of the number of diseased plants and roots.

TABLE 3

Results of inoculating different varieties of sweet potatoes on Arlington Farm, June 16, 1914

VARIETY	NUMBER INOCULATED[1]	NUMBER INFECTED JULY 1	NUMBER INFECTED JULY 7	NUMBER INFECTED JULY 15	OCT. 14[2] NUMBER PLANTS DEAD OF THOSE MARKED INFECTED JULY 15	NUMBER DISEASED	PLANTS RECOVERED[3]	TOTAL NUMBER NOT INFECTED AT ANY TIME DURING SEASON	ROOTS INFECTED	TOTAL PLANTS DISEASED DURING SEASON	PER CENT OF INFECTION
Red Jersey	34	22	28	31	28	4	2	0	0	34	100
Nancy Hall	33	26	27	31	28	3	0	2	1	31	93.9
Gen. Grant Vineless	38	29	29	29	13	18	3	4	0	34	89.4
Key West Yam	43	25	26	30	27	8	1	7	1	36	83.7
Eclipse Sugar Yam	26	6	7	13	9	11	1	6	0	21	80.7
Yellow Yam	42	16	17	22	18	7	1	16	1	26	61.9
Vineless Yam	29	9	11	14	9	7	1	12	1	17	58.6
Southern Queen	46	16	16	16	10	15	2	19	0	27	58.6
Georgia	47	6	12	20	14	11	0	22	1	25	53.1
Florida	29	10	10	10	3	10	1	15	0	14	48.2
Yellow Strassburg	44	8	8	8	1	17	1	25	0	19	43.1
Miles Yam	35	1	1	2	2	12	0	21	0	13	37.1
White Yam	38	9	10	10	4	6	1	27	0	11	36.8
Pierson	43	5	5	5	0	14	0	29	0	14	32.5
Red Brazilian	39	3	4	5	1	7	2	29	0	10	25.6
Dahoney	39	3	3	3	0	2	2	35	0	4	10.2

[1] Ten checks were used for each variety. These plants were punctured at the base of stem in the same way as those inoculated. None became infected.

[2] Plants were dug and final records were made on October 14.

[3] This shows the number of plants which either recovered from the disease or where the old vine died and new healthy sprouts formed.

in the South, such as Red Bermuda and Florida, scarcely make potatoes in northern states.

Certain varieties of sweet potatoes have been grown continuously in some regions for many years. This continuous use of one variety has enabled the farmers to establish a good market for their crop. For example, the Yellow Jersey, grown largely in New Jersey and Delaware, finds a ready market in the large cities of the East. These markets do not willingly take the yam types grown extensively in the South. The growers therefore would lose a profitable industry even though the southern types might thrive well, or, at least, would be compelled to build up a new market for their crop. In the South where the yam types are mostly produced a different market has been established, and one which prefers those types to any others. In fact, the markets preferring the southern sweet potatoes do not like the Yellow Jersey or similar varieties and will not take them if others can be secured. Where such local conditions are met with the only apparent solution of the problem appears to be the establishment of a resistant strain by selection.

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OBSERVATIONS RELATIVE TO AN OBSCURE GRAPE AFFECTION

F. E. Gladwin

With One Figure in the Text

During the summer of 1910 the attention of the writer was attracted to the sickly appearance of the leaves of several Concord vines growing in a young vineyard 3-years-set, on the experimental grounds of the New York Agricultural Experiment Station at Fredonia. In the year 1911 the trouble again appeared in this vineyard and also in others 6-and-7-years-set respectively. In 1912 the affection was present in all the vineyards mentioned, and in addition, in one 23-years-set and located on a very different soil type from the others.

In 1913 the trouble was again present in all the vineyards except in the 23-years-old one, and in addition in two others that were set in 1910. In 1914 the same condition appeared with the exception of the oldest vineyard as noted for the season of 1913. Of the five years in which this disease has been noted, that of 1912 is the one in which the trouble was the most serious.

At its first appearance the disease was associated with Chlorosis of the old world, but close observations, and examinations of the literature pertaining to this disease tend to show that the two are not due to the same causes. Limestone soils are not common in the Chautauqua Grape Belt, and in fact the soils on which these vineyards are located are deficient in lime. Thus far the addition of lime to these soils has not influenced the affection either by increasing or lessening it.

The leaf blight disease described by Fairchild[1] while having some symptoms and environmental factors in common, differs from the one under discussion in several material points. He states that leaf blight is common only to young vines 3 to 4-years-set. Our observations show, that under certain conditions which will later be described, the present trouble can be found on much older vines.

Again he says that "the worst attacks of the disease occur upon cold, heavy soils containing a large percentage of clay and rich in nitrogenous matters." It is true that four of the vineyards under discussion are situ-

[1] Fairchild, D. G. Diseases of the grape in western New York. Jour. Myc. 6: 95–98. 1891.

ated upon heavy soil types yet they are deficient in nitrogen, as has been shown by chemical analyses and the response to applications of stable manure and commercial nitrogen. One of the vineyards, the one 23-years-set, and in which the disease was observed in severe form, in only one year 1912, is planted on a loose gravelly loam of an approximate depth of 20 feet. Another is on a light clay to silt loam and well drained.

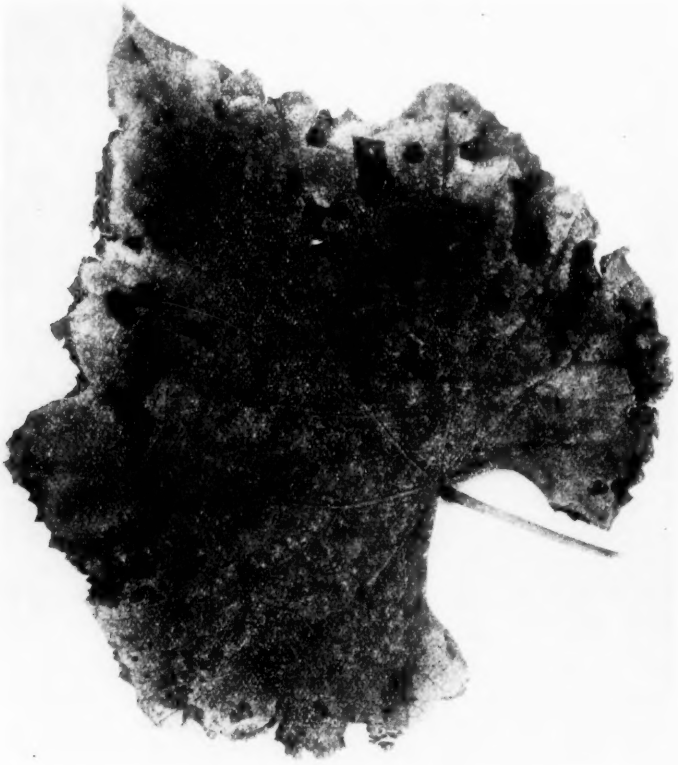

FIG. 1. Grape leaf showing typical marginal and intervenular blighted areas.

In some respects the symptoms manifested by the vines later described, are similar to those of the California vine disease of Vinifera varieties, discussed by Pierce,[2] yet the two are so different in other particulars that they can not be attributed to the same causes. Pierce states that cuttings taken from vines affected with the California vine disease carry with them the disease and thus it is perpetuated. Cuttings taken from

[2] Pierce, N. B. Grape diseases on the Pacific Coast. U. S. Dept. Agr., Farmers' Bul. 30: 7–14. 1895.

vines with the affection under discussion have not, after three years growth, shown that they carry the sickness, but are perfectly normal in top and root.

During the dormant season there are no external evidences that would indicate the trouble, except that wood growth is not as well matured nor is it so great as on the unaffected vines. However early in July in average seasons the apical leaves of affected shoots show a streaked yellowing in the intervenular spaces. The vineyardist but rarely observes this, the beginning stage. A little later other leaves on the shoots develop a pallidness. The discoloration is more marked near the margin and eventually the pallid areas coalesce, and form a yellowed band extending around the margin. As the season advances this band of tissue dries and becomes functionless. The deadened area is further increased by a drying out of the intervenular tissue, extending from the margin inward to the midrib until in extreme cases the entire tissue of the leaf, except that along the main veins becomes brown and dead. Often only one side of the leaf shows this extreme stage; in other cases, after the margin is dead, isolated and scattered spots of dead tissue develop, without order, over the blade, and these may coalesce so that dead areas of considerable size are produced quite apart from those at the margin. A leaf that has gone through the successive stages of dying back from the browning of the margin, and subsequent killing inward of intervenular tissue, has only narrow strips of green living tissue immediately adjacent to the principal veins and veinlets; all other tissue is brown or black. When the entire leaf is affected it often becomes curled.

During the first year an affected vine may be diseased in but few shoots from a single cane, or at most on two or three canes. The year following additional canes and shoots may show the trouble until the whole vine is affected. It appears to be cumulative. Vines have been observed that were completely defoliated some time previous to the period of maturity. The fruit of affected vines does not color nor is the normal amount of sugar fixed. The berries do not acquire full size and in extreme cases shell from the cluster. When the vine is only moderately affected shelling may not follow but the fruit is not attractive nor palatable.

The wood growth is checked materially, and such as there is, does not mature properly, so that much of it is winter killed. That not killed is soft and light in weight and apparently deficient in stored plant food. The difficulty of obtaining good fruiting wood for the succeeding year becomes a serious problem.

As the appearance of this sick condition was first associated with the Chlorosis of the Vinifera in France, the accepted remedial measures of that country were tried, namely the application of sulphate of iron to the

soil, and with water, as a spray. Three years experiments along these lines failed to yield beneficial results.

That the trouble is connected in some way with the relationship of roots to the soil is evident from the fact, that those vines observed as the worst affected, extended diagonally across a knoll, and in each row the vines were affected for only a short distance either side of it. The soil over this knoll, both soil and subsoil, was compared with that from other parts of the same vineyard, and where the vines were unaffected. That over the knoll on which the sick vines were located, proved to be of much finer texture, of a more silty nature and more deficient in organic matter than in other parts. Chemical analyses of the two revealed no striking differences in nitrogen, phosphorus, potassium or calcium carbonate content. In two other vineyards the subsoil is very similar to that just described and the trouble was proportionate to the percentage of yellow silt present. In the case of the 23-years-old vineyard these soil differences were not seen. In a part of another young vineyard the subsoil was black silt. Here the soil remained wet throughout the growing season by reason of underground seepage water. However considering all the diseased vines, the greater percentage was located on yellow silt soil.

Thus far applications of nitrogen in nitrate of soda and dried blood, potassium in the form of sulphate and muriate of potash, phosphorus in acid phosphate and basic slag, sodium sulphate, calcium oxid or calcium carbonate have not affected the degree of the affection.

In 1912, the year that this trouble was observed in the 23-years-old vineyard, located on the deep gravelly loam soil already mentioned, the first valuable data were obtained as to the probable influence that favored the sickly condition. Since 1909 four plats of two rows each, have been sown, during late July each year, to a green manure crop of mammoth clover. This was plowed under the following spring. In 1913 it was decided to allow the clover in two of these plats, one limed the other not, to grow during the summer and to mow at intervals, leaving the mowings as a mulch. The summer of 1912 was very dry. For twenty-seven days previous to July 13 no rain occurred. On this day 0.41 inches fell. Some days previous to this rainfall the vines in the clover mulched plats began showing symptoms of disease and this developed into the worst case that had yet been seen. Vines in a depression of the rows were not so badly affected as were those on the slopes at either side. The rows immediately bounding the clover rows showed negligible amounts of the disease and there was practically none in the remainder of the vineyard. No differences were discernible between the limed and unlimed plats, while the vines in the two other plats in which clover had been plowed under in the spring were healthy. The fruit from the diseased clover plats was char-

acteristic of that examined from the other vineyards in previous years. In 1913 these vines showed the effect of the trouble from the previous year, but in 1914 they were normal healthy vines and equal to the others.

Considering the data at hand it would seem that a lack of moisture in the soil or the inability of the vines previously affected to absorb the optimum amount is the underlying cause of the trouble. The yellow silt soil, being deficient in organic matter, is not able to retain the moisture in sufficient amounts to balance the loss through transpiration, and the loss through evaporation. Especially is this true in extreme drouths. It has been mentioned earlier that the affection appears to be cumulative and this would naturally follow, as the leaves become functionless, fibrous growth of the root system is checked, hence lessened absorbing surface for the following year. In the case of the vines of the clover plats, the fact that the trouble was not observed previous to 1912 and that it has not recurred would indicate that the growing clover roots utilized the soil moisture to the detriment of the vine, while the amount of the mulch was not sufficient to check evaporation from the surface. Had the season not been one of drouth, it would seem consistent to believe no such ill effects would have been noted. The fact that the affection was considerably less in a depression where the water accumulated would tend to bear out this belief.

In the vineyard located on black silt, fed by subterranean springs over which the soil was wet throughout the summer, it is believed that excessive moisture prevented the development of root systems commensurate with the needs above ground. Examination of the root system of affected vines as compared with nearby healthy ones of the same age and variety showed a marked deficiency of fibrous roots in the former, and also a number of main roots already dead. It is known that vines growing on wet soils do not make a normal amount of root growth. With this vineyard lack of aeration may contribute to the trouble. This land was recently tile drained and while there is a steady discharge throughout the summer, a large part of it is probably due to the interception of an underground water course. In later years when the soil becomes more porous as a result of the tiling, the affection should diminish, if it be due to a curtailment of moisture supplied to the aerial parts.

Reports and observations of other vineyards indicate that the trouble is not uncommon. Invariably it is found under situations similar to those mentioned. Also it is more common with young vines. This latter can be accounted for from the fact that the root system of newly planted vines are not extensive enough to afford ample absorbing surface. Older vines with deeply penetrating roots, hence are able to better withstand drouth. Any practice that results in the accumulation of humus in the soil suggests

itself as a possible relief. With soils that do not allow sufficient root development, by reason of wetness, thorough tile drainage seems to be the logical solution. Under such conditions a certain amount of injury will result for several years but the degree will depend to a considerable extent upon the summer rainfall and the thoroughness with which this is conserved. Conservative pruning will in considerable degree relieve, through lessening the amount of fruit to be matured.

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NOTES ON SOME DISEASES OF TREES IN OUR NATIONAL FORESTS. V

GEORGE GRANT HEDGCOCK

This paper is a continuation of notes[1] on forest tree diseases based on observations and collections made chiefly by the writer, assisted by Dr. E. P. Meinecke, Dr. Jas. R. Weir, Dr. Perley Spaulding, Mr. W. H. Long, Mr. Carl Hartley, Mr. G. F. Gravatt, and Mr. J. F. Rogers, of the Office of Investigations in Forest Pathology during a survey of the diseases in the forests of the United States.

SPECIES OF RAZOUMOFSKYA (ARCEUTHOBIUM) ATTACKING GYMNOSPERMS

1. *Razoumofskya pusilla* (Peck) Kuntze occurs on the following species in the northeastern United States: it very commonly attacks the black spruce, *Picea mariana*,[2] and less frequently the white spruce,[2] *Picea canadensis*, the red spruce, *Picea rubens*, and the eastern larch, *Larix americana*. A larger form of Razoumofskya, which probably does not belong to this species, is described by Engelmann[3] as *Arceuthobium douglasii* var. *microcarpa*. This form occasionally is found on the Engelmann spruce, *Picea engelmanni*, in Idaho, Colorado, Arizona, and New Mexico, the blue spruce, *Picea parryana*, in New Mexico, and the weeping spruce, *Picea breweriana* in California.

2. *Razoumofskya douglassii* (Engelm.) Kuntze very commonly attacks the Douglas fir, *Pseudotsuga taxifolia*, throughout the range of this tree from the Rocky Mountains to the Pacific Coast, and from the Mexican boundary to the Dominion of Canada.

3. *Razoumofskya americana* (Nutt.) Kuntze is very common on the lodge-pole pine, *Pinus contorta*, throughout the range of this species of tree, especially in the central and northern Rocky Mountain region and on the eastern slopes of the Sierra Nevadas in California and Oregon.

[1] Notes on some diseases of trees in our national forests: I, Printed privately at Washington, D. C., February, 1914; II, Phytopathology **2:** 73-80. April, 1912; III, Phytopathology **3:** 111–114. April, 1913; IV, Phytopathology **4:** 181–188. June, 1914.

[2] The nomenclature for trees in this paper is that of Geo. B. Sudworth, U. S. Dept. Agr., Bur. For. Bul. 17. 1898, and Forest Trees of the Pacific Slope, For. Serv. 1908.

[3] U. S. Geol. Surv. west of 100th Merid. (Wheeler's Rept.) Bot. **6:** 253. 1878.

4. *Razoumofskya cryptopoda* (Engelm.) Coville the eastern form of the following species is very common on the western Yellow pines, *Pinus ponderosa*, and *Pinus ponderosa scopulorum*, in the states in the region of the Great Basin and Rocky Mountains from eastern Washington to New Mexico. In New Mexico and Arizona, this mistletoe or a closely related species, attacks the Chihuahua pine, *Pinus chihuahuana*, and Mayr's pine, *Pinus mayriana*.

5. *Razoumofskya campylopoda* (Engelm.) Piper occurs very commonly on *Pinus ponderosa* in the Pacific Coast region. It or closely related species attacks Jeffrey's pine, *Pinus jeffreyi*, the Monterey pine, *Pinus radiata*, the Sabine pine, *Pinus sabiniana*, and Coulter's pine, *Pinus coulteri*, in California.

6. *Razoumofskya divaricata* (Engelm.) Coville occurs frequently on the nut pines. *Pinus edulis* is attacked from Colorado, Utah, and California southward to Mexico, and *Pinus monophylla* in Arizona and California. The knob-cone pine, *Pinus attenuata*, is occasionally attacked in California by a form of Razoumofskya which may belong here.

7. *Razoumofskya laricis* Piper often attacks the western larch, *Larix occidentalis*, in Montana, Idaho, Washington, and Oregon. It is occasionally found on Lyall's larch, *Larix laricina*, in Idaho.

8. *Razoumofskya tsugensis* Rosend, is frequently found in the northwestern United States from Montana to Washington on the western hemlock, *Tsuga heterophylla*, and occasionally in Washington on Merten's hemlock, *Tsuga mertensiana*.

9. *Razoumofskya douglasii abietina* (Engelm.) Piper, the smaller form of dwarf mistletoe on species of Abies, occasionally attacks the cork fir, *Abies arizonica*, in Arizona and New Mexico. It is common on the white fir, *Abies concolor*, from Arizona northward to Oregon; on the grand fir, *Abies grandis*, from California to Washington and Idaho; on the red fir, *Abies magnifica*, and on the Shasta fir, *Abies shastensis*, in California; on the noble fir, *Abies nobilis*, and the Amabalis fir, *Abies amabilis*, in Washington; and on the alpine fir, *Abies lasiocarpa*, from Colorado to Montana.

10. *Razoumofskya occidentalis abietina* (Engelm.) Coville, the larger form of dwarf mistletoe on species of Abies, is frequently found in Utah, California and Oregon on *Abies concolor;* and in California and Oregon on *Abies grandis* and *Abies magnifica*.

11. *Razoumofskya cyanocarpa* (A. Nelson) Rydberg, occasionally attacks the limber pine, *Pinus flexilis*, from Colorado to Montana; the white-barked pine, *Pinus albicaulis*, in Idaho and Montana; and the bristle-cone pine, *Pinus aristata*, in Utah and California. This mistletoe, or a closely related species, attacks the mountain white pine, *Pinus monticola*, in California; the sugar pine, *Pinus lambertiana*, in California and Oregon; and the Mexican white pine, *Pinus strobiformis*, in Arizona.

THE EFFECT OF PLANTS OF SPECIES OF RAZOUMOFSKYA ON THEIR HOSTS

All these species of Razoumofskya retard the growth of the trees they attack. This results from an excessive growth of the portion of the tree attacked whether it be a twig, a limb, or the trunk itself. The immediate effect on the host in the region of a primary infection by Razoumofskya is to cause hypertrophy in the cambium of the wood tissues which form in the vicinity of the sinkers or roots of the young mistletoe plants. This produces usually a fusiform swelling. Later the spread of the sinkers of the mistletoe throughout the branches of an affected limb stimulates an increased production of twigs, and the formation of a witches broom. The form of this broom varies with the habit of growth of the limbs and twigs of species of tree attacked. These witches brooms often develop to an immense size, and at the same time their increased vigor decreases that of all other portions of the tree. This materially lessens the annual increment to the wood in the trunk.

Some species of dwarf mistletoes in the northwestern United States, for example, *Razoumofskya laricis*, and *R. tsugensis*, form dense witches brooms, composed of very brittle limbs and twigs. The weight of heavy snows on the brooms, cause them to break, usually near the point of attachment. This lessens the amount of foliage on the trees and directly retards their growth. At the same time a wound is made at the point of rupture which enables heartrotting fungi, such as *Trametes pini* (Brot.) Fr. and *Echinodontium tinctorium*, E. & E. to gain an entrance into the heartwood of the limb of the trees. These two species of fungi alone are responsible for an enormous loss in values in our older forests in the western United States.

Plants of these species of Razoumofskya or dwarf mistletoes are so abundant in many localities that from 25 to 50 per cent of the trees are attacked. The resulting loss in tree growth is consequently enormous, and the problem of eradicating these mistletoes becomes one of prime importance in forest hygiene.

SPECIES OF PHORADENDRON ATTACKING ANGIOSPERMS

1. *Phoradendron flavescens* Nutt., a polymorphic species, attacks many broad-leaved trees, and occasionally shrubs, in the eastern, southeastern and southern United States. It has several quite distinct forms. The following are the principal ones: a large obovate-leaved form, with the largest white berries of any species, is common on red maple, star-leaved gum, black locust, black walnut, elm, and other species in the eastern, southeastern, and southern United States; an orbicular-leaved form with smaller whitish berries occurs chiefly on oaks in the southern United

States; a narrow-leaved form with white berries is found in Florida on oaks, ash and other species.

Phoradendron flavescens in its eastern forms has been either reported or collected on the following host species: *Acacia roemeriana, Acer rubrum, A. saccharinum, Aesculus glabra, Betula nigra, Benzoin aestivalis, Castanea dentata, Celtis occidentalis, C. mississipiensis, Crataegus* sp., *Diospyros virginiana, Fagus atropunicea, Fraxinus americana, F. berlandieriana, F. caroliniana, Hicoria cordifolia. H. pecan, H. glabra, H. ovata, Gleditsia aquatica, G. triacanthos, Juglans nigra, Liquidambar styraciflua, Melia azedarach, Nyssa sylvatica, Papyrus papyrifera, Platanus occidentalis, Populus deltoides, Prosopis juliflora, Prunus angustifolia, P. cerasus, P. serotina, Pyrus communis, P. malus, Quercus alba, Q. brevifolia, Q. catesbii, Q. chapmani, Q. digitata, Q. georgiana, Q. lyrata, Q. macrocarpa, Q. marilandica Q. minor, Q. nigra, Q palustris, Q. phellos, Q. texana, Q. velutina, Q. virginiana, Robinia pseudacacia, Salix amygdaloides, Salix nigra, Sapindus marginatus, Sophora secundiflora, Toxylon pomiferum, Tecoma radicans, Tilia floridana, Ulmus americana, U. crassifolia, U. pubescens,* and *Xanthoxylum clava-herculis.* The oak and ash trees in southeastern United States are often attacked by the narrow-leaved form. From Oklahoma to Texas the orbicular or round-leaved form, *Phoradendron flavescens orbiculatum* Engelm., is common on oaks.

2. *Phoradendron flavescens macrophyllum* Engelm. is represented in Arizona and New Mexico by two or three large-leaved forms with three- to five-nerved leaves and with white berries which are smaller than those of *Phoradendron flavescens* on gum, maple, and other trees in the eastern United States. It attacks the following species of trees: *Alnus oblongifolia, Fraxinus americana, F. cuspidata, F. velutina, Juglans rupestris, Platanus wrightii, Populus angustifolia, P. deltoides, P. fremontii, Prunus* sp., *Prosopis odorata, Pyrus communis, P. malus, Robinia pseudacacia, Salix nigra, S. taxifolia,* and *Sapindus marginatus.*

A smaller, round-leaved form with small greenish-white berries, related to *Phoradendron flavescens orbiculatum* Engelm., occurs in Arizona, New Mexico, and extreme western Texas. It has been either collected or reported on the following trees: *Quercus arizonica, Q. chrysolepis, Q. emoryii, Q. gambelii, Q. hypoleuca, Q. oblongifolia, Q. reticulata,* and *Q. toumeyii.*

In California and Oregon there is a somewhat variable form of *Phoradendron flavescens,* which has large leaves, usually five-nerved, and greenish-white to white berries. This form corresponds to *Phoradendron flavescens macrophyllum* in Arizona and New Mexico but probably is not identical with it. This form has been noted in Califorina on the following host species: *Alnus rhombifolia, Platanus racemosa, Populus trichocarpa, P. nigra, Pyrus communis, P. malus, Robinia pseudacacia,* and *Salix nigra.*

3. *Phoradendron villosum* Nutt. is a species found in California and Oregon, which is related to *Phoradendron flavescens* but distinct from it. It has more or less rounded leaves, small white or pink berries, and is related to *Phoradendron flavescens orbiculatum* Engelm. It is known to attack most commonly various species of oaks. It has either been collected or reported on the following host species: *Aesculus californica, Arctostaphylos manzanita, Forestessin neo-mexicana, Quercus agrifolia, Q. californica, Q. chrysolepsis, Q. douglasii, Q. garryana, Q. lobata, Q. wislizeni,* and *Rhus diversiloba.*

4. *Phoradendron californicum* Nutt., a leafless species with long stems and pink berries, attacks a number of species of trees in the southwestern United States from Texas to California. It has been either collected or reported on the following host species: *Acacia greggii, Cercidium torreyanum, Condolia spathulata, Covillea tridentata, Dalea spinosa, Olneya tesota, Parkinsonia microphylla, Prosopis juliflora,* and *P. odorata.*

SPECIES OF PHORADENDRON ATTACKING GYMNOSPERMS

1. *Phoradendron pauciflorum,* Torr., a polymorphic species which has also been called *Phoradendron bolleanum* Eichl. is found in the southwestern and western United States. There are at least two distinct forms of this mistletoe. All forms have pink berries, and leaves varying in shape from spatulate to linear. A form, evidently a distinct species with linear, villose leaves, attacks species of Juniper. It has been found on *Juniperus occidentalis,* and *J. utahensis.* Three spatulate-leave forms of Phoradendron which evidently differ in species from the previous are known, one on *Abies concolor,* a second on *Cupressus arizonica, C. glabra* and *C. macnabiana,* and a third on *Juniperus californica, J. monosperma, J. occidentalis,* and *J. pachyphloea.* Dr. Meinecke reports a white-berried, spatulate-leaved form on *Cupressus sargentii.*

2. *Phoradendron juniperinum* Engelm. attacks nearly all species of Junipers in the southwestern and western United States, ranging from New Mexico and Colorado to California and Oregon. The leaves of this species are mere scales and its berries are pink to pinkish-yellow. It attacks *Juniperus californica, J. monosperma, J. occidentalis, J. pachyphloea, J. scopulorum,* and *J. utahensis.* It has been reported on *Cupressus arizonica* and *Pinus monophylla,* but neither the writer nor any other member of this Office has seen specimens of this species of Phoradendron on Cupressus or Pinus. This species also attacks *Libocedrus decurrens* in California and Oregon, on which host it is known as *Phoradendron juniperinum libocedri* Engelm., but the writer doubts if this is a good variety because it is similar to the form on Juniperus.

THE EFFECT OF PLANTS OF SPECIES OF PHORADENDRON ON THEIR HOSTS

One or more species of Phoradendron may be found in great abundance in many localities in the southern, southwestern, and western United States. The loss due to the effects of Phoradendron in lessening tree-growth in the United States is less than that caused by Razoumofskya, nevertheless it is apt to be underestimated.

The increased chlorophyll-bearing surface in the leaves and stems of plants of species of Phoradendron, naturally makes them less dependent on the host for a supply of carbohydrates, and as a result they must exert a less deleterious effect, since they require chiefly a supply of water and mineral foods. In this regard they might be compared to plants rooted in soil. On the other hand the water they receive is in the form of sap from the host and it naturally follows that it contains in addition to water all the necessary food constituents required by the host. The Phoradendron plant although it does build up carbo-hydrates, apparently uses some of the supply furnished by the host. The amount used by the parasite depends upon the relative amount of chlorophyll-bearing surface it possesses. This may be relatively large as in forms of *Phoradendron flavescens*, or relatively small as in case of *Phoradendron californicum*, and *P. juniperinum*, where the chlorophyll is confined almost exclusively to the stems.

Plants of species of Phoradendron exert a stimulus upon the cambium tissues of the host at the point of their attachment. This frequently causes very marked hypertrophy locally. These hypertrophied areas or burls are especially prominent in trees of *Abies concolor* when attacked by *Phoradendron pauciflorum*. The grain of the wood in these burls or galls is much distorted, and as a result the tensile strength of the wood is lessened. Finally the tree is often broken off by winds at the point where these burls are formed. Burls are found to some extent in case of other conifers attacked by Phoradendron.

In case of species of Abies, Cupressus and Juniperus, it is often noted that the Phoradendron plant diverts the growth of the limb at the point of attack, so much that the limb and its branches are starved and killed from this point outward. This leaves the limb without branches, and in their stead a living Phoradendron plant borne at the resulting apex. In Arizona and New Mexico, Juniper trees are often found where *Phoradendron juniperinum* has taken complete possession of a number of the more important limbs of the tree. In all such cases the growth of the host either is almost entirely checked or greatly retarded. Similar instances occur in trees of many broad-leaved species when attacked by *Phoradendron californicum*, *P. villosum*, and *P. flavescens* or its varieties.

In no instance has it been observed that Phoradendron plants exert a beneficial effect on the host, but on the contrary the injury caused by their presence appears to be in direct proportion to the relative increase in number and size of the Phoradendron plants on any given tree. This has been noted especially in case of ornamental and shade trees in cities and towns of the southern and western United States.

The injurious effect of plants of species of Phoradendron and Razoumofskya on trees can no longer be doubted. Apart from sentiment, there can be no reason for permitting them to remain on shade and ornamental trees, and in the forests all well-matured plans for scientific forest management should include the cutting of all trees diseased by mistletoes whenever practicable, especially when an area is lumbered. Where seed trees are left in such areas, care should be taken to select those that are not diseased by mistletoes in order to prevent the spread of these parasites to the trees of the younger generations of the forest.

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A NEW MACROPHOMA ON GALLS OF POPULUS TRICHOCARPA

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WITH THREE FIGURES IN THE TEXT

In the fall of 1909 a collection of very noticeable galls occurring on *Populus trichocarpa* Torr. & Gr. was made with the supposition that they were caused by an insect, *Saperda populnea* L. Upon examination, the galls showed no evidence of insect origin as was supposed, and sections were made to determine, if possible, the cause of the hypertrophy. The first attempt along this line proved fruitless, for too little attention was paid the condition of the bark. In March, 1910, another collection was made for further study.

Thin transverse sections of the galls taken from the older twigs of an infected tree and stained with eosin disclosed large subcarbonous, pycnidia in great numbers embedded in the cortex. These pycnidia were filled with numerous, elongate, hyaline spores borne on short simple sporophores arising from a mycelial layer directly above the hypothecium. These spores filled the cavity of the fruiting body and escaped through the ostiole in coiled, thread-like masses. Upon placing a few of the spores in sugar solution and in a solution of the extract from Populus twigs, it was found that they germinated readily in both media, producing hyphae similar to those which were found extending downward from the pycnidia into the host tissue. It might be well to mention here that the pycnidia were not found upon the normal stems, branches or twigs.

Subsequently, experiments were started to determine whether the fungus in question caused the hypertrophy of the twigs and branches or whether the abnormal growth could be traced to other causes. An attempt was also made to locate the ascogenous stage of the parasite. So far, examinations of old galls have revealed nothing but the pycnidial stage.

The fungus was identified as a species of Macrophoma but its specific identity could not be determined.

In November, 1914, samples of the galls were sent to Dr. C. L. Shear, of the Bureau of Plant Industry, who determined the fungus to be an undescribed species of Macrophoma and named it *Macrophoma tumefaciens* n. sp. Shear's description of the cottonwood-gall fungus follows:

Macrophoma tumefaciens Shear n. sp.

Pycnidia numerous, depressed, globose, buried in the cortex, subcarbonous, irregular in shape, slightly erumpent, subepidermal, 120 to 350 μ in diameter; ostiole slightly elevated, non-papillate; pore small, dark-colored; pycnospores elongate, subellipsoid to subrhomboid, frequently irregular in outline and inequilateral, mostly obtuse, hyaline to pale lemon yellow, 24 to 40 $\mu \times 7.5$ to 12 μ, ejected in coiled thread-like masses, contents granu-

FIG. 1. Populus trichocarpa, showing the numerous galls upon branches and twigs. Photograph reproduced by courtesy of J. R. Weir.

lar; germinating readily and producing dark-brown, thick-walled hyphae; sporophores 6 to 12 μ long, simple, filamentous; hyphae dark brown, septate, 10 to 12 μ wide; cell wall 1 to 1.5 μ thick.

Habitat: On gall-like swellings of branches and twigs, *Populus trichocarpa*, Missoula, Montana, November, 1914, Ernest E. Hubert.

Type specimen: No. 2808, Pathological Collections, Bureau of Plant Industry, Washington, D. C.

Co-type No. 121Y in the collection of E. E. Hubert.

Cross inoculations and cultural experiments are well under way but no definite results can be given at present. A later paper is intended to be published dealing with the pathological relations of the fungus to the host, and an attempt will be made to produce in cultures or to locate in nature the perfect stage.

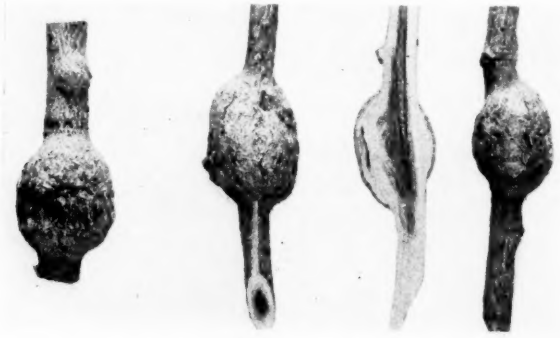

FIG. 2. Galls from Populus trichocarpa. The specimen at the left shows a young gall forming just above an old one. Reduced one-half.

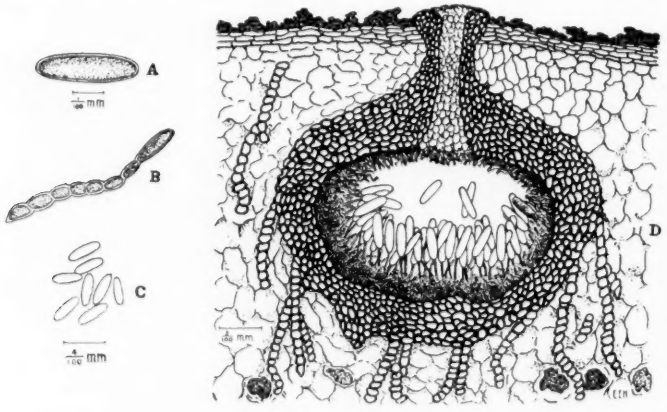

FIG. 3. Macrophoma tumefaciens. *a*, pycnospore showing granular contents; *b*, pycnospore germinating; *c*, pycnospores showing variations in size; *d*, pycnidium embedded in tissue of Populus trichocarpa gall. Note hyphae extending downward into tissue of host.

These galls with the accompanying fungus are widely distributed through Montana, occurring principally upon twigs and branches of *Populus trichocarpa*. Collections and observations were made in Montana at Missoula, Ralston, Philipsburg, Florence, Drummond, Custer, Bearmouth, Deerlodge, and Wisdom; in Idaho, at Eagle, Avery, and St. Maries.

The galls appear almost invariably at the point where the twigs and branches fork. Those occurring at the base of young twigs have a tendency to cause that portion of the twig, in advance of the gall, to become dwarfed and finally to die. The galls are so numerous on some of the older trees that a greater portion of the lower branches appear to have been killed by them. Young trees in the near vicinity of an old infected tree also bear the same galls upon twigs and branches and show evidence of the injurious presence of these growths.

Since the gall formation can not be traced to insect work, it is reasonable to suppose that the hypertrophy is caused by the hyphae of the fungus, *Macrophoma tumefaciens*, extending into the growing tissues and stimulating them to unusual activity. At any rate the injury to the infected tree is great enough to merit investigation as to the primal cause of the disorder.

MISSOULA
MONTANA

A PRELIMINARY STUDY OF ERGOT OF WILD RICE

FAITH FYLES

WITH PLATE XI

In my experiments with the cultivation of Canadian wild rice, *Zizania aquatica* L. & Z. *palustris* L., I had often noticed ergot among the seed. A search of the literature revealed only one record of the occurrence of ergot on wild rice (R. H. Denniston (1)).

The questions arose, first, is this ergot identical with the typical ergot (*Spermoedia clavus*(D. C.) Fries), of rye, second, will cereals and other grasses growing in its neighborhood be in danger of infection by it?

Dr. Robert Staeger (2) of Bern, Switzerland, who has been successfully experimenting with ergot of rye for many years, has already proved that ergot, which cannot be distinguished morphologically, may, however, require quite a different range of host plants. He has shown by a series of successful experiments that ergot of rye, *Secale cereale*, will infect the following grasses:

Anthoxanthum odoratum, *Hierochloe odorata*, *Poa pratensis*, *Phalaris arundinacea*, *Arrhenatherum elatius*, *Dactylis glomerata*, *Poa sudetica*, *Festuca pratensis*, *Festuca arundinacea*, *Barley*, *Hordeum murinum*, *Alopecurus pratensis*, *Briza media*, *Poa hybrida*, *Poa compressa*, *Calamagrostis arundinacea*, *Bromus sterilis*, *Secale cereale*, Spanischer Doppelroggen.

He has further shown that *Claviceps microcephala* Tul., (now *Spermoedia microcephala* (Wallr.) Seaver) of which the typical host plant is *Phragmites communis* Trin., does not exist on a single host plant subject to attacks of *Spermoedia clavus* (D. C.) Fries. *S. microcephala* infects, he states, besides its host, *Nardus stricta*, *Molinia coerulea*, and *Aira caespitosa*.

I secured a small quantity of fresh wild rice seed, gathered at Treadwell, Ontario, September 29, 1913. Among the seed were many ergots, several of which were still within the glumes of the wild rice. On October 9, 1913, most of these sclerotia were placed in pots of sand outside the laboratory window. Occasionally they were watered until they became buried in snow. Others of the same lot of sclerotia were placed in sterilized sand and water in two stender dishes kept in the laboratory at some distance from the window. They were given water twice, once in November and once in December. On December 16, two sclerotia had already germinated. One sclerotium had 19 stromata from 1 to 5 mm. high and the

other showed 11 stromata. Judging from later observations, germination must have taken place a week or ten days before, i.e., a little over two months after the sclerotia were gathered. Although these ergots had been kept at room temperature from September 29, they had already, in the open air, been subjected to 2° frost on September 16, 1913.

The ostiola of the perithecia appeared in the form of minute dots on December 22 when the stromata were only 7 mm. high and the heads 1 mm. in diameter. They gradually became more distinct as the perithecia protruded until finally the stromata looked like diminutive spiked clubs. The first spores were seen on December 30. There were 40 stromata at one time on this sclerotium, the most mature being 2.5 cm. high with a head 2.5 mm. wide, while the youngest could just be seen through a crack in the cortex. One other ergot of this sowing produced 40 stromata and a third 37. The remainder did not germinate so freely, the last showing only 10 heads. Of later sowings, the greatest number of heads on a single intact ergot was 48, and the least 11. Broken pieces of ergots produced from 3 to 7 stromata. The heads were light buff in color with lavender stalks, becoming deeper and slightly reddish with age. At the height of maturity, the perithecial chambers measured 250 to 325 μ in length and 150 to 160 μ in width. The asci were 200 to 215 μ long and about 4 μ wide. The spores were 150 to 180 μ long.

On reading H. T. Güssow's account (3) of tri-septate spores recorded in his ergot of barley, I examined the spores of my fungus and am able to confirm his finding of three septa in each spore, which evidently indicates a generic character.

As wild rice is essentially an aquatic plant and naturally most of the ergots would drop into the water to float away or be washed ashore, more sowings were made: In unsterilized sand and water; unsterilized mud and water and in distilled water alone. In the first experiment, the sclerotia germinated abundantly and produced strong and healthy heads. Those in mud and water with about 2 mm. of water above the mud flourished also, and grew 4.5 to 5 cm. high with heads 2.5 mm. wide. Those floating on the distilled water germinated freely on all sides of the sclerotia, but their growth was slow, and, when they were only 5 mm. high, they became covered with mould fungi. The mould was removed as far as possible, and the sclerotia were transferred to sterilized sand and water. A few of these stromata finally produced spores, but most of them perished before maturity.

In all these sowings, most of the ergots were placed merely on top of the sand or mud. A few were buried an inch or more, but not one of the latter germinated. Even those which were brought to the surface after being buried three months did not show signs of life.

On May 22, 1914, the last of the number of sclerotia, which had been left exposed all winter outside were divided; one-half was brought into the laboratory and the other half left in a box of mud and water beside the growing wild rice. On June 30, when those in the laboratory had almost finished their spore-shedding and were 4 to 4.5 cm. high, those outside had just begun to shed their spores. This was before the rice had shown any signs of blooming. The growth was very thick and short—only 1 cm. high with heads 3 to 3.5 mm. in diameter and of a dark reddish purple color, indicating the strong positive heliotropism to which Claviceps is known to be subject.

The following table gives the data of the different stages of growth taken from one or two sclerotia of each sowing; all harvested in September, 1913.

INFECTION EXPERIMENTS WITH ERGOT OF WILD RICE

At the time of the first sowing of ergot of wild rice, October 9, 1913, we had just received a small barrel of wet wild rice seed of the autumn's gathering. In this were found a dozen seeds or more that had already germinated. As this autumnal germination of wild rice is very unusual, the young seedlings were carefully planted in large glass jars with 15 cm. of mud. The jars were then filled with water and placed, some in the laboratory and some in the greenhouse for infection experiments. Later, twelve pots of rye and barley were sown for the same purpose. But the wild rice grew slowly in the winter and the barley and rye did not make the desired progress. It was necessary to make repeated sowings of ergot all through the winter in order to have on hand mature stromata when the grasses bloomed. When, at last, the first stalks of rye and barley were in bloom, the spore-shedding of one entire lot of ergot was over and the next lot was not yet mature.

Unfortunately, at that time, I had not read Dr. Staeger's (4) recent work on infection experiments with ergot of rye, in which he showed that the conidia still adhering to the wintered sclerotia of ergot of rye were capable of germination and infection even after ten months' rest. On reading this paper, I at once re-examined the ergot, which had been kept dry in a stoppered glass jar, and found several ergots with a whitish coating, some of which were still within the glumes of the wild rice. The glumes were carefully removed and a dry white substance was found at the base of the ergots as well as inside the glumes. These ergots and glumes were soaked in distilled water for a few minutes. The water at once became milky; a drop under the microscope proved it to be full of conidia.

TABLE 1

Data on the development of sclerotia harvested September 1913

DATE OF SOWING	SOWN IN	FIRST APPEARANCE OF STROMATA	FIRST APPEARANCE OF PERITHECIA	HEIGHT AT THAT TIME	FIRST EMERGENCE OF SPORES	HEIGHT AT THAT TIME	NO. OF STROMATA ON THE SCLEROTIUM	GREATEST HEIGHT OF STROMATA
1913		1913		*millimeters*		*millimeters*		*millimeters*
Oct. 9	Sterilized sand and water	Dec. 10	Dec. 22	7	Dec. 30	20	40	25
Oct. 9	Sterilized sand and water	Dec. 18	Dec. 22	10	Dec. 31	29	37	30
Oct. 9	Sterilized sand and water	Dec. 18	Dec. 23	12	Jan. 2	20	40	23
		1914						
Oct. 9	Sterilized sand and water	Feb. 4	Feb. 14	15	Feb. 20	25	15	28
Oct. 9	Sterilized sand and water	Feb. 4	Feb. 14	15	Feb. 26	23	10	23
		1913						
Dec. 16	Sterilized sand and water	Dec. 23	Jan. 2	10	Jan. 7	12	27	15
Dec. 16	Sterilized sand and water	Dec. 23	Jan. 2	10	Jan. 8	12	23	20
Dec. 16	Sterilized sand and water	Dec. 29	Jan. 11	15	Jan. 15	20	25	21
		1914						
Dec 16	Sterilized sand and water	Mar. 6	Mar. 16	8	Mar. 24	10	26	15
Dec. 16	Sterilized sand and water	Mar. 6	Mar. 18	12	Mar. 28	14	25	21
Dec. 17	Unsterilized mud and water	Jan. 5	Jan. 13	8	Jan. 19	20	36	45
Dec. 17	Unsterilized mud and water	Jan. 5	Jan. 20	13	Jan. 25	27	29	50
		1913						
Dec. 17	Unsterilized sand and water	Dec. 31	Jan. 13	5	Jan. 22	24	22	40
		1914						
Jan. 3	Sterilized sand and water	Feb. 2	Feb. 13	10	Feb. 19	15	38	20
Jan. 3	Sterilized sand and water	Feb. 4	Feb. 16	11	Feb. 26	17	15	24
Feb. 20	Sterilized sand and water	Mar. 14	Mar. 31	10	Apr. 14	17	48	20
Feb. 20	Sterilized sand and water	Mar. 17	Mar. 31	9	Apr. 13	15	34	18
Feb. 20	Sterilized sand and water	Mar. 18	Apr. 2	9	Apr. 11	10	46	15
Mar. 28	Sterilized sand and water	Apr. 14	Apr. 30	10	May 4	13	14	10
Mar. 28	Sterilized sand and water	Apr. 17	May 2	10	May 6	14	11	17
May 4	Sterilized sand and water	May 23	June 2	10	June 10	23	40	30
May 4	Sterilized sand and water	May 25	June 3	10	June 11	23	40	30
May 4	Sterilized sand and water	May 27	June 3	10	June 12	25	23	35

First inoculation of barley and rye

The barley and rye spikes, which were then at anthesis as well as those a little past this stage, and others in which the kernel had begun to form, were all inoculated with this spore-containing solution on April 24, 1914. No honeydew appeared on any of them, not even after a month. Only healthy seeds were seen on the now ripened heads.

First inoculation of wild rice

In the meantime, the wild rice in the greenhouse had bloomed. In the afternoon of May 11, fresh ascospores of the ergot were caught in water and sprinkled on the pistillate flowers of one stalk of wild rice, pollinated that morning. On May 15, the flowers were still very fresh, and, fearing they had been inoculated too soon and determined to obtain some positive result from this experiment, I re-inoculated them, both with fresh ascospores and with conidia obtained from the wintered ergots. Thus, when honeydew did appear, it was uncertain whether the conidia or ascospores were the cause of it. On the morning of May 30, 1914, two large drops of honeydew were found on this stalk of wild rice. A little of it in a drop of water was examined under the microscope, and found to be full of conidia. They measured 9 to 12 μ in length and 2.5 to 3.5 μ in width. (Those of *S. clavus* are 7 μ in length and 3.5 μ in width.)

On June 1, there were four drops of honeydew. The lowest drop, which was the first to appear, had changed somewhat in appearance, being now less clear and more viscid. On June 3, there were unmistakable signs of two purplish sclerotia. June 6, honeydew had ceased oozing, and three large and firm sclerotia were seen. June 11, as the sclerotia and also the plant seemed to be drying prematurely, the stalk bearing the ergots was cut off to preserve it. A little of the fresh honeydew had previously been secured in a small bottle.

Second inoculation of wild rice

On June 1, 1914, a second jar of wild rice in another wing of the greenhouse bloomed for the first time. This was inoculated with the spore-containing water of the dry ergots of September 1913.

On June 11, a drop of honeydew appeared on the uppermost flower and in two days the beginnings of a sclerotium were visible. On June 17, a lower flower also showed honeydew. The success of this second infection experiment with wild rice, which had only been inoculated with last year's conidia, proves that the conidia are still capable of germination even after nine months' rest, which confirms Dr. Staeger's discovery in regard to the ergot of rye.

EXPERIMENTS WITH RYE AND OTHER HOSTS OF ERGOT OF RYE

The rye plants, which were inoculated at the same time as the wild rice, i.e., May 11, 1914, with fresh ascospores and May 15, with old conidia, still showed no signs of honeydew.

On May 30, when honeydew first appeared on wild rice, a little of it was used to inoculate the last pot of rye in bloom. With some more of this same fresh honeydew, on June 6, the following grasses, which had been brought in from the experimental plots outside before they had bloomed, were inoculated. *Poa pratensis*, *Dactylis glomerata* and *Alopecurus pratensis*, which are all hosts of the ergot of rye. On June 8, rye growing out in the open, and on June 10 and 11 *Arrhenatherum elatius* and more rye in the greenhouse, also brought in from outside, were inoculated with the spore-containing solution of the sclerotia of September 1913, but without any positive result.

On June 12, thirty-five stalks of rye in bloom were inoculated with old conidia—with negative results.

July 3, three more stalks of rye then in bloom were treated with fresh conidia, but without any appearance of honeydew.

A number of oats in flower, also specimens of *Elymus dasystachys* and *Agropyron tenerum* were inoculated repeatedly with old and fresh conidia without any positive result.

It may be said that the study of this form of ergot proved most interesting. The rapidity of the development of stromata and the large number observed make this ergot a most desirable one for class purposes. From certain morphological differences, the difference in the measurements of conidia and ascospores, the fact that plants readily infected with spores of *S. clavus* (D. C.) Fries, proved immune in my experiments, and from some other biological features, I feel justified in considering this ergot a distinct species, and in proposing for it a new specific name. On completion of my work, the usual diagnosis will accompany the final account.

In conclusion, I wish to acknowledge my indebtedness to H. T. Güssow, the Dominion Botanist, for his kindness in editing the manuscript and making timely and important suggestions.

DIVISION OF BOTANY
EXPERIMENTAL FARM
OTTAWA, CANADA

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EXPLANATION OF PLATE XI

Artificially infected flowers of wild rice.
Various stages of germinating ergots.

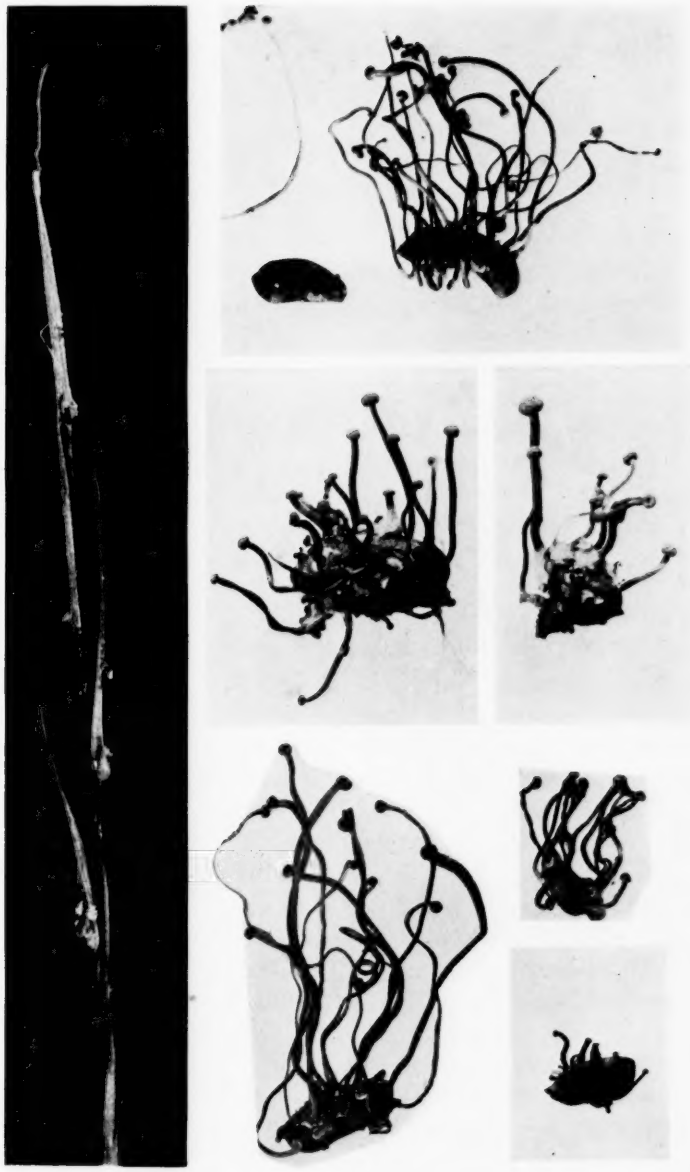

Photo. A. E. Kellett.

A POWDERY MILDEW ON CITRUS

C. N. Carter

With Plate XII and One Figure in the Text

In May 1911 a fungus was found by the writer upon a Dancy tangerine tree (*Citrus nobilis*, Lour. var.) in an orange grove at Duarte, Los Angeles County, in the southern part of California. The fungus appeared to be a powdery mildew and specimens were sent to Prof. W. T. Horne of the University of California, at Berkeley, for examination. The writer was advised that the fungus was undoubtedly a powdery mildew and was worthy of further study. In the same year inoculations were made upon roses and citrus trees other than tangerines near the original infestation. Conidia from tangerine leaves were placed on rose leaves and conidia from the rose leaves were placed on marked leaves of the tangerine, but in all cases results were negative. A covering was placed over the leaves in every case.

In 1912 the mildew was first observed in June, and the writer began to search the immediate neighborhood and nearby districts for its further occurrence. It was found on a tree of Dancy tangerine growing alone about two miles from the point of original observation. A grove of large and particularly good tangerine trees near Azusa was examined and the mildew found rather abundantly. While not enough to appreciably affect the vigor of the trees, it immediately suggested the possibility of more serious development such as has occurred with the powdery mildew of apples in some parts of California.

The leaves are the only parts of the tree affected. The young, tender growth with leaves about two thirds developed is most susceptible. The first appearance of the fungus on the young leaf occurs on the edge, usually as isolated spots 2 to 5 mm. broad. The hyphae of the mycelium can be plainly distinguished as very fine radiating lines. There is a darkening and usually a perceptible depression of the invaded leaf surface, followed by a general buckling of the whole leaf. The crumpling increases and new infection may take place along the center, but always on the upper surface of the leaf. On closer observation it will be found that the coloring matter has disappeared and in the older stages the spots become decidedly yellow. The old affected leaves which remain on the tree have a more or less characteristic appearance. The edges turn down very sharply, especially where the infection has taken place on the edges of the leaves.

A favorable place for older infections of the mildew seems to be along the midrib. The greatest number of affected leaves are to be found on the north and west sides of the tree, towards the center and near the ground. In the center of the tree, where it is more or less shady, all stages of the mildew may be found. Very seldom do affected leaves occur near the top of the tree.

It is evident that mist and fog favor the spread and growth of the fungus. Apparently the most favorable conditions are furnished by decidedly damp mornings when the sun does not come out until midday. Although the past spring was exceptionally cool and cloudy, the mildew fungus did not grow so vigorously as in the previous seasons when there were more moist foggy mornings.

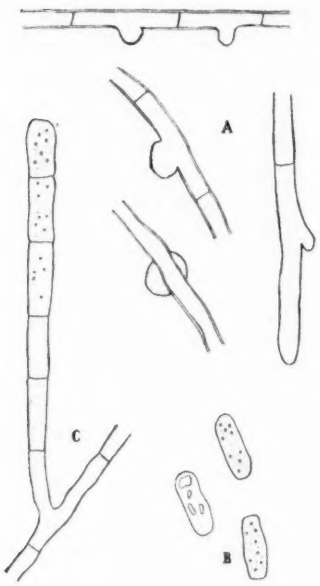

FIG. 1. *a*, Appresoria on hyphae of the mycelium; *b*, a group of three conidia drawn from dried material; *c*, conidiophore with chain of conidia. All magnified 333.3 diameters.

The mycelium may be readily seen spreading out on the leaf as superficial, white threads, radiating more or less and sparse at first. In subsequent stages the mycelium becomes denser but never felty. The patches spread out and finally become confluent, especially along the midrib of the leaf. The conidiospores are visible to the naked eye, standing up from the leaf. It would appear that the mycelium which is barren late in the season remains dormant on the green leaves through the winter and when favorable conditions return produces conidia to infect the younger leaves in the spring. No perithecia have been observed.

The mildew on *Citrus nobilis* presents the microscopic characters shown in figure 1. The mycelium measures 4.5 to 6.7 μ in diameter. The conidiophores are 60 to 120 μ long and about 12 μ broad at the top, becoming slightly narrower below. The conidia are borne in chains of four to eight. In making measurements of twenty or more conidia from dried specimens, considerable variation in size was noted. They are usually barrel-shaped with flattened or slightly rounded ends and measure 20 to 28 μ by 10 to 15 μ. The appresoria are roundish and are 10 to 12 μ in diameter.

In order to find out whether such an Oidium has been reported on *Citrus nobilis*, W. T. Horne, of the University of California requested

Mrs. Flora W. Patterson of the Bureau of Plant Industry, United States Department of Agriculture, Washington, D. C., to have search made of the mycological literature. The work was carried out by Mr. F. J. Veihmeyer and no Oidium was found which corresponds in any way with the one under discussion.

"*Oospora citri-aurantii* (Ferr.) Sacc. et Sydow, *Oidium Citri-aurantii* Ferraris, Malpighia XIII, p. 12, has been reported as occurring upon putrefying fruit of *Citrus aurantium* in Italy."

"*Acrosporium fasciculata*, Greville's *Flora Edinensis*, is reported on decaying oranges. This is presumably the same fungus as *Oidium fasciculatum* Berk. and *Oospora fasciculata* (Berk.) Sacc. and Vogl."

These are the only two cases of Oidium reported from the thorough search made, and in both cases it occurred on decaying fruit and therefore is not to be confused with the fungus under consideration.

Accordingly the name *Oidium tingitaninum* is proposed for it in reference to its particular host, the tangerine, and the following technical diagnosis, based on a study of both living and dried specimens is given.

Oidium tingitaninum sp. nov.

Patches developing on immature leaves, epiphyllous near the margins and along the center becoming more or less confluent and sometimes covering most of the leaf, whitish, sparsely powdery, the leaf surface below the mycelium early becoming stained brown, darkening with age and finally losing part of its green color. Mycelium not dense nor felty, mostly disappearing with age; hyphae 4.5 to 6.7 μ in diameter. Appresoria rounded; conidiophores 60 to 120 by 12 μ; conidia in chains of four to eight, colorless, finely granular, barrel-shaped with slightly rounded ends, variable in size 20 to 28 by 10 to 15 μ.

Habitat: On leaves of Dancy tangerine, *Citrus nobilis* Lour, var. Los Angeles County, California. Type material has been deposited in the Herbarium of the University of California, at Washington, D. C. and in the New York Botanical Gardens.

Latin diagnosis. O. maculis in foliis nondum maturis orientibus, epiphyllis, atque margines et aream centralem foliorum occupantibus, deinde plus minusve confluentibus nunc maximam superficiem foliorum vestientibus, albidis, sparse pulverulentis, superficie foliorum sub maculis fere ab initio fuscedecolorata sed aetate provecta fusciore et ultime partem coloris viridis amittente; mycelio neque denso neque dense lanoso intertexto, aetate provecta maxime evanescenti; hyphis 4.5–6.7 μ in diametro; appressoriis rotundatis; conidiophoris 60–120 x 12 μ; conidiis 4–8 catenatis, hyalinis, minute granulatis, doliiformibus, apicibus parve rotundatis, in magnitudine variabilibus, 20–28 x 10–15 μ.

SUMMARY

A powdery mildew disease due to a species of Oidium has been observed on the leaves of the Dancy tangerine, *Citrus nobilis* Lour. var. This is believed to be the first report of a disease of this type on plants of the genus Citrus.

The fungus concerned has been found only in a limited area in southern California. It appears to be restricted to one variety of tangerine orange and to depend on the occurrence of warm weather, wet fog, and abundant new growth at the same time. It has been under observation for four seasons and there is no reason to fear that it will have any economic importance in California.

The name *Oidium tingitaninum* is suggested for this fungus and a technical diagnosis is presented.

A number of inoculations have been made but these have not succeeded on tangerine or any other plants. Oidiums from other plants have failed to grow on tangerine.

From observations made in the field during more than three years, the fungus reappears about the month of May, but there is considerable variation from year to year as to its abundance.

I am under obligations to Prof. W. T. Horne for his special interest and attention in this work. Professor Setchell very kindly prepared the Latin diagnosis and gave suggestions as to the proper name. I am indebted to Mr. C. O. Smith for his interest in the work and to the Slauson Ranch, Azusa, for material. Mr. R. M. Teague of the San Dimas Citrus Nurseries very kindly supplied budded citrus stock for inoculation work.

COLLEGE OF AGRICULTURE
UNIVERSITY OF CALIFORNIA
BERKELEY, CALIF.

EXPLANATION OF PLATE XII

FIG. 1. Tangerine leaf shortly after reaching full size and yet not fully mature. The most conspicuous spots of Oidium show on the midrib.

FIG. 2. Leaf at very nearly the same stage as in figure 1. Spots here are more strongly developed toward the margin.

FIG. 3. Six tangerine leaves at full maturity showing the characteristic buckling due to powdery mildew. The mycelium was still evident on these leaves but was beginning to disappear. All nearly natural size.

CARTER: POWDERY MILDEW ON CITRUS

PHYTOPATHOLOGICAL NOTES

A new scarlet oak disease. A new fungus belonging to the genus Botryodiplodia has been found to be causing a serious killing of the young branches on scarlet oak in the vicinity of Cincinnati. About 150 to 200 scarlet oaks, *Quercus coccinea,* are affected at the Spring Grove cemetery. Further investigations will be carried on this coming summer and complete results published in the near future. Material of similar oak diseases will be appreciated. D. C. BABCOCK

OHIO AGRICULTURAL EXPERIMENT STATION

Lightning injury to onions. Since reading the article in a recent issue of PHYTOPATHOLOGY on Lightning Injury to Potato and Cotton Plants by Jones and Gilbert, the writer thinks it of sufficient interest to make note of a case of lightning injury which came to his attention in an onion field at Marion, New York, in the summer of 1914. In this case the injury to the onion plants, which were about one foot high, was noticed shortly after a severe electrical storm, and it was observed by workmen that the lightning struck in the locality where injury was later discovered. The field was visited by the writer some days later when it was found that the plants were killed over an area of about eight feet in diameter, while plants immediately adjoining this area showed injury. The muck soil in the center of the lightning-struck spot was disturbed or thrown out to a depth of approximately four inches and over an area of a few inches.

M. T. MUNN

Correction. In the April number of PHYTOPATHOLOGY (p. 97), in connection with the discussion of lightning injury to potatoes, I cite Prof. F. C. Stewart as authorizing the statement that he attributed to lightning an injury observed in New York potato fields. This is incorrect. My statement was based upon what proves to be a misinterpretation of Professor's Stewart's joking reference to the "lightning bug" in a letter he wrote me after reading the abstract of the same paper in the program of the Philadelphia meeting. The joke was really a good one, but, as with some of my earlier experiences with lightning flashes, I was not quick enough to interpret it correctly. The facts are, as Professor Stewart intended me to understand from the first letter and now authorizes me to state on his behalf, that he considers the evidence to justify the conclusion that the "lightning bug" responsible for the damage in the New York potato fields referred to is the stalk borer, *Papaipema nitela* Guen. While he did not

find the insects he did find evidence of the larval invasion in every dead plant examined.

It is noteworthy that the general appearances of the dead spots in the potato fields were so similar in the two cases and this emphasizes the need of attention that the two types of injury be not confused.

L. R. JONES

Personals. Dr. F. Kølpin Ravn, Professor of Plant Pathology at the Royal Landbohøjskolens, Copenhagen, Denmark, will come to this country during the first week in May and engage in a series of conferences with officials of the United States Department of Agriculture and of state experiment stations in the various states, on problems concerned with cereal cultivation, particularly cereal diseases. It will be remembered that Doctor Ravn has published particularly valuable papers on the leaf diseases of barley.

Dr. J. Rosenbaum, until recently Instructor in Physiology in Cornell University, has been appointed to a position as Assistant Pathologist in Truck Crop Disease Investigations, Bureau of Plant Industry, United States Department of Agriculture, to take up work on potato diseases.

Dr. F. L. Stevens has prepared a preliminary draft of a Bibliographic Appendix to his book The Fungi which Cause Plant Diseases, consisting of 32 typewritten pages of additional references to literature. Copies may be secured from Dr. Stevens at Urbana, Illinois, for 50 cents each.

A short course on the diseases of plants as related to their keeping quality during transportation and storage was given by the University of Illinois, February 8 to 20, by Dr. F. L. Stevens and Mr. Geo. L. Peltier.

Mr. Ernest E. Hubert, a graduate of the University of Montana, and former employee of the Forest Service of the Department of Agriculture, has been appointed to a temporary position as Field Assistant in Forest Pathology in that Department and stationed at Missoula, Montana, where he will work under the direction of Dr. Jas. R. Weir during the period from January 1 to July 1.

The death is reported of Mr. Y. Takahashi, Pathologist of the Hokkaido Agricultural Experiment Station, Japan, on November 14, 1914. The position has been filled by the appointment of Mr. Leiya Ito, Assistant Professor at the Tohoku Imperial University, and Pathologist of the Experiment Station at Sapporo, Japan.

Publications of the Insecticide and Fungicide Board. The attention of pathologists is called to the publications issued by the Federal Insecticide and Fungicide Board of the United States Department of Agriculture, portions of which may be of interest. The Service and Regulatory Announcements, eight numbers of which have been issued, the first dated March 16, 1914, and the last April 3, 1915, contain extracts from various letters written by the Board to manufacturers of insecticides and fungi-

cides, expressing opinions of the Board with reference to the labeling of insecticides and fungicides in accordance with the Insecticide Act of 1910. These announcements are prepared primarily for and distributed among manufacturers, the object being to more fully acquaint them with the views of the Board in regard to the manner in which various types of insecticides and fungicides should be labeled, and thereby enable them to avoid errors, which they would be likely to make if such information were not available. For example, in S.R.A. No. 1, pages 6 and 7, are found extracts from letters written to certain manufacturers concerning faulty recommendations on the labels of Bordeaux mixtures, which contain the opinion of the Board in regard to certain statements representing very common types of errors. This publication also contains the notices of judgment of cases under the National Insecticide Act which have been settled in court, some of which may be of interest to plant pathologists. The Insecticide Decisions, six of which have been issued, the first August 26, 1911, and the last February 12, 1912, represent another form of attempt by the Board to enable manufacturers to label their products in accordance with the Act. Insecticide Decision No. 6, relative to inert and active ingredients of Bordeaux mixture, is of this nature.

M. B. WAITE
ERRETT WALLACE

Desiderata. Announcements will be inserted in the Phytopathological Notes section of this JOURNAL, concerning the wants of members of the Society in the way of cultures, specimens, or exchanges.

San Francisco Meeting of the American Phytopathological Society. The Society at its Philadelphia meeting decided to hold a special meeting at San Francisco, August 2–7, in connection with the meeting of the American Association for the Advancement of Science.

It is hoped that there will be as good an attendance as possible. All members planning to visit the Pacific Coast or the Exposition should arrange to do so at the time of this meeting. An interesting program is expected. Special attention will be given to Pacific Coast problems.

C. L. SHEAR, *Secretary-Treasurer*

The Western American Phytopathological Society. A meeting of the Western American Phytopathological Society was held at Corvallis, Oregon, on December 29 and 30, 1914. The first meeting was held on the afternoon of December 29 and was given over to discussion of topics of special interest to members present. Considerable part of the afternoon was spent in the discussion of pear blight in the Pacific Coast region. The visiting members were entertained at a banquet in the evening and on the following day three sessions were held at which the following program was presented:

Investigation of physiological plant diseases.....R. E. SMITH
Wilt disease of carnations.....C. J. WIGHT

Sappy bark disease of apples and Polystictus versicolor, WM. T. HORNE AND C. J. RODGERS
Melaxuma of walnuts ..... H. S. FAWCETT
Preliminary studies of resistance of Prunus to inoculation with Bacterium tumefaciens C. O. SMITH
A new filbert disease in Oregon ..... H. P. BARSS
Notes, observations and investigations of plant diseases in Western Oregon ... H. L. REES
Inoculation studies with apple tree anthracnose ..... H. S. JACKSON
Minor notes on interesting diseases ..... H. S. JACKSON
Warnock's tree paint as a cure for fire blight ..... J. W. HOTSON
Internal brown streak of potato ..... S. S. ROGERS
Cottony rot of lemon (Sclerotinia libertiana) ..... C. O. SMITH
The prevention of wood decay in trees ..... WM. T. HORNE
Apple scab spraying experiments in the Hood River valley ..... J. R. WINSTON

A telegram of greetings was sent to the parent society which was in session at the same time in Philadelphia.

The society voted to hold the next meeting at San Francisco and the executive committee was instructed to arrange for a joint meeting with the parent society.

About twenty-five persons attended the meeting, including representatives from Oregon, Washington, California and British Columbia.

WM. T. HORNE, *Secretary-Treasurer*

Charles Edwin Bessey. It is with the deepest regret that we announce the death of Prof. Charles E. Bessey, the distinguished botanist and teacher, and a beloved charter member of The American Phytopathological Society. Dr. Bessey died at his home in Lincoln, Nebraska, February 25, in the seventieth year of his age, having been born May 21, 1845. He was Professor of Botany in the University of Nebraska for thirty-one years. He leaves a wife and two sons, Carl and Ernst.

A brief biographical sketch of Professor Bessey prepared by the writer will be found in the Asa Gray Bulletin, Volume 4, page 23, March, 1896.

Though not a specialist in Plant Pathology, Professor Bessey took an active interest in the subject and was ready and willing at all times to do what he could to promote its interests and those of our Society. Only a short time before he was taken sick he expressed to the writer his belief that we should be able to secure an endowment for PHYTOPATHOLOGY, and also his determination to render all the assistance he could to accomplish this purpose. The loss to botany caused by Dr. Bessey's death will be deeply felt by his fellow botanists. How great this loss is, not only to science but to humanity, can only be understood by those who had the good fortune to sit at his feet as students or have enjoyed association with him as a friend and co-worker. His "boys and girls," as he affectionately termed his students who specialized in botany, will feel that they have lost not only a noble and devoted teacher, but also a beloved friend and adviser.

C. L. SHEAR

LITERATURE ON AMERICAN PLANT DISEASES[1]

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———A Gymnosporangium with repeating spores. (Abstract.) Phytopathology **4,** no. 6: 408. December, 1914.

———How to use aecium and similar terms. (Abstract.) Science n. s. **41,** no. 1048: 172. January 29, 1915.

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[1] This list aims to include the publications of North and South America, the West India Islands, and islands controlled by the United States, and articles by American writers appearing in foreign journals.

All authors are urged to coöperate in making the list complete by sending their separates and by making corrections and additions, and especially by calling attention to meritorious articles published outside of regular journals. Reprints or correspondence should be addressed to Miss E. R. Oberly, Librarian, Bureau of Plant Industry, U. S. Dept. Agric., Washington, D. C.

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"Only claim to originality in this bulletin is in its arrangement and presentation with some few additional comments by the author which have not heretofore appeared, and some colored illustrations. . . "

São Paulo. Secretaria da Agricultura, Commercio e Obras Publicas. Cancro das batatas inglezas. Bol. Agr. [São Paulo] **15**, no. 4/5: 390–391. Abril/Maio; no. 8/10: 701–704, illus., Agosto/Outubro. 1914.

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